Anxiety and Its Disorders

The Nature and Treatment of Anxiety and Panic

Second Edition

David H. Barlow

The Guilford Press
New York London

A Division of Guilford Publications, Inc.
72 Spring Street, New York, NY 10012
www.guilford.com

Printed in the United States of America

This book is printed on acid-free paper.

Last digit is print number: 9 8 7 6 5 4 3 2 1

Library of Congress Cataloging-in-Publication Data

Barlow, David H.
Anxiety and its disorders : the nature and treatment of anxiety and panic / David H. Barlow.—2nd ed.
p. ; cm.
Includes bibliographical references and index.
ISBN 1-57230-430-8
1. Anxiety. I. Title.
[DNLM: 1. Anxiety Disorders. WM 172 B258a 2002]
RC531 .B28 2002
616.82'223—dc21 2001040754

CHAPTER 12

Posttraumatic Stress Disorder

TERENCE M. KEANE
DAVID H. BARLOW

One of the great questions in all of psychopathology concerns etiology. It is widely agreed that determining what causes emotional and behavioral disorders may ultimately lead to the development of preventive interventions that reduce the likelihood of a disorder's ever occurring. For this reason, the study of posttraumatic stress disorder (PTSD) is critically important in the study of psychopathology in general. With PTSD, it is possible to specify the onset of psychological and behavioral disturbance with good precision. As a result, it is a condition that may prove to be scientifically invaluable as we attempt to understand the relative contributions of constitutional factors, psychological parameters, and environmental contributants to the development of psychopathology.

Among the most important questions addressed in the past 20 years of research on PTSD is why some people develop this disabling psychological condition in the wake of exposure to traumatic life events when others, seemingly exposed to the same event, do not. To explain this, one needs to examine many different factors that make one person more vulnerable to developing PTSD following a particular traumatic stressor. Does rape "cause" PTSD? Does combat "cause" PTSD? The literature to date suggests that although these environmental events contribute to the development of PTSD, they generally interact with other factors to yield PTSD. To state it more accurately, we know the proximal or precipitating events (e.g., rape, combat) that activate this disorder in vulnerable individuals, but these events may not be sufficient to lead to the disorder in all exposed individuals. However, as the events become more severe in nature, the chances that they will lead to PTSD in people who experience them increase. As Sutker and Allain (1996) eloquently stated in their evaluation of the impact of torture experienced by U.S. former prisoners of war: "An accumulating literature suggests that as trauma events become universally brutal; more horrific, gruesome, and prolonged; and more threatening to life, the greater the likelihood that negative sequelae will develop. . . . Eventually all victims succumb to psychological distress." As the severity of a traumatic event increases, in other words, the role of personal or individual risk factors diminishes. For less severe (although still traumatic) events, constitutional, psychological, and environmental factors will all interact to determine who will and who will not develop PTSD. It is clear that PTSD or other negative psychological outcomes can arise in even the most emotionally stable and

healthy individuals if the stressor is severe enough. The sudden death of a child constitutes one such stressor; prolonged torture represents another.

PTSD is characterized by high levels of anxiety, panic, and often depression. Yet the symptoms that appear to distinguish PTSD from other psychological disorders are the reliving experiences, such as a preoccupation with, nightmares about, and flashbacks recapitulating the precipitating event. These symptoms are typically present with an emotionally numb state, avoidance of cues that are reminders of the event, and a range of signs of elevated anxiety (such as insomnia, poor concentration, heightened startle reactions, hypervigilance for danger, and irritability).

When the disorder was initially conceptualized in the *Diagnostic and Statistical Manual of Mental Disorders*, third edition (DSM-III), traumatic events were considered extreme life stressors that were outside the range of normal human experience (American Psychiatric Association, 1980). However, several outstanding epidemiological studies since that time have yielded high prevalence rates of exposure to traumatic events in the general population, dismissing the idea that traumatic events were "outside the range of normal human experience." In contemporary society, traumatic events are frequent across age, race, gender, and socioeconomic status (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995).

HISTORY AND BACKGROUND

Historical depictions of PTSD can be traced to the story of Ulysses in Homer's *Iliad* and *Odyssey*, which are among the oldest literature in Western civilization (Shay, 1992). The devastating impact of fighting the Trojan War resulted in damaged character among many of those who survived. These changes were observed to be long-standing in nature and resulted in difficulties in returning to the warriors' homeland. Remarkably, the symptoms and behaviors described in Homer's works are similar to those observed among individuals with PTSD today.

Stephen Crane's description in *The Red Badge of Courage* of the psychological effects of combat experience in the American Civil War resulted in the term "soldier's heart." The effects of war were seen as a combination of the losses sustained in the war with the removal of soldiers from the interpersonal connections associated with their families, friends, and communities. Thus the adverse psychological consequences of combat, and of war more broadly, have been recognized for hundreds if not thousands of years.

Scientific study of war's effects reached new heights with World War II and its aftermath. Studies of combatants, former prisoners of war, survivors of the concentration camps and death camps, and resistance fighters indicated significant levels of psychological distress and mood disturbance among those who survived (e.g., Grinker & Spiegel, 1945). Although these conditions were not yet considered to be PTSD, they set the stage for the ultimate recognition of the long-term negative effects of exposure to traumatic stressors. This would, in time, lead to the construction of the PTSD diagnosis. Terms such as "shell shock," "combat fatigue," and "war neurosis" all seemed to capture the essence of war-related PTSD, though there was as yet no appreciation for how other traumatic life stressors produced the same psychological effects. It wasn't until Ann Burgess, working at Boston City Hospital, observed the impact of rape on women that the term "rape trauma syndrome" was coined (Burgess & Holmstrom, 1974). Concomitantly, American and Australian veterans of the Vietnam War were also searching for an understanding of their psychological distress following that politically difficult conflict. Advocates for these two disenfranchised populations (i.e., rape survivors and Vietnam veterans) teamed with scholars

who had studied and treated World War II veterans and concentration camp survivors to create the synergy necessary to construct the diagnosis of PTSD.

Yet even before the great wars of the 20th century, anxiety as a response to severe stress was observed and labeled "traumatic neurosis" by Oppenheim (1892; cited by Kraepelin, 1896) or *Schreckneurose* (i.e., "fright neurosis") by Kraepelin (1896). Kraepelin considered this condition a separate clinical entity "composed of multiple nervous and psychic phenomenon arising as a result of severe emotional upheaval or sudden fright which build up great anxiety; it can therefore be observed after serious accidents and injuries, particularly fires, railway derailments, or collisions, etc." (Kraepelin, 1896; translation by Jablensky, 1985, p. 737). In view of the early and clear recognition of this emotional disorder, it is surprising that controversy regarding its validity emerged during the 1970s and 1980s (Figley, 1978; Goodwin & Guze, 1984). Although much of the initial controversy about PTSD's validity has ceased (Keane, Wolfe, & Taylor, 1987), the nature of the disorder still instigates considerable debate in the field (Yehuda & McFarlane, 1995).

Epidemiological studies now confirm that PTSD occurs following a wide range of extreme life events. War, rape, torture, crime, vehicular accidents, industrial accidents, sudden death of a loved one—all result in the development of PTSD in a certain proportion of those exposed. Many early examples of these reactions have been recorded over the centuries by those inclined to write them down. One of the better-known descriptions is the reaction of the famous 17th-century diarist, Samuel Pepys, after the Great Fire of London in 1666. This catastrophe resulted in substantial loss of life and property and in marked disorganization in the city, all of which was very well described by Pepys. Fully 6 months after the fire, he recorded: "It is strange to think how to this very day I cannot sleep a night without great terrors of fire; and this very night could not sleep to almost 2 in the morning through thoughts of fire" (quoted in Daly, 1983, p. 66). Insomnia, and recurring dreams of the event, are of course prominent features of PTSD as we know it today; however, Pepys also manifested mild depersonalization, as well as some characteristic guilt about saving himself and his property while others died (known as "survivors' guilt").

In addition to recurrent and intrusive recollections and dreams concerning the event, an individual may experience "flashback" episodes wherein he or she seems to experience a recurrence of at least a portion of the traumatic event. Individuals describe these experiences "as if it's happening all over again." These flashbacks can often contain input from all senses—sights, sounds, smells, tastes, and tactile sensations recapitulating the traumatic event—and they can be extraordinarily frightening.

Extreme distress and avoidance of cues or reminders of the event also accompany PTSD. This avoidance may involve an inability to remember aspects of the traumatic event itself. The debate over the nature of trauma memories has captured the attention of clinicians and cognitive psychologists the world over. The discussion hinges on the notion of "recovered memories," which are typically (but not exclusively) of child sexual abuse. Rancorous at times, this debate led the International Society for Traumatic Stress Studies to impanel a representative team of clinicians, researchers and scholars to study the matter. The senior editors of this effort (Roth & Friedman, 1998) concluded as follows, based on the available scientific evidence:

> We know that people forget childhood traumas and that this is not limited to people in treatment or to people whose trauma is sexual abuse. We also know that people can accurately recall memories of documented childhood trauma that they report having previously forgotten,

> and that a wide range of triggers seem to be associated with these memories. Most memory recovery appears to be precipitated in situations that include cues that are similar to the original trauma and does not occur as a direct result of psychotherapy (i.e. memories implanted or imposed by a therapist). However, it is possible, and indeed many would argue likely, that therapists who fail to conform to accepted standards of practice may promote a "recovered memory" of an event that never occurred. (p. 23)

The issue of recovered memory has unfortunately been tied to the societal problems associated with childhood sexual abuse. Yet there is considerable clinical evidence across different types of traumatic events that individuals store memories for traumatic events in unusual ways. For example, news about an explosion aboard a U.S. aircraft carrier brought a World War II Navy veteran to seek counseling from the National Center for PTSD for a traumatic event he had experienced 45 years earlier, which he reportedly had not thought about since his return stateside. When the newscasts of the current explosion reminded him of the death and destruction that he experienced as a function of a Japanese kamikaze attack, he developed acute symptoms of PTSD. Did he forget this event? Did he distract himself effectively for nearly half a century? Did he take this opportunity to reveal the experience to others? This is the nature of the scientific debate and discussion regarding traumatic memory and what has been termed "recovered memory" of trauma. More scientific information is needed before firm conclusions can be drawn regarding the nature of recovered memories of traumatic events.

Additional symptoms of PTSD also include emotional numbing, described by patients as an inability to feel any positive emotions such as love, contentment, satisfaction, or happiness. As such, the emotional numbing is disruptive to intimate and interpersonal relationships. Conceptualizations of emotional numbing are rare, but Litz and his colleagues (Flack, Litz, Hsieh, Kaloupek, & Keane, 2000; Litz, 1992; Litz & Keane, 1989; Litz, Orsillo, Kaloupek, & Weathers, 2000) have approached this problem from an information-processing perspective. Employing experimental psychopathology paradigms and using epidemiological data, they have concluded that the intensity of the anxiety and arousal symptoms in patients with PTSD appears to drive the extent of the emotional numbing observed.

PTSD is further characterized by trouble sleeping, trouble concentrating, an enhanced startle reaction, and difficulty controlling anger. Hypervigilance for danger is yet another component of the condition. Finally, individuals with PTSD live for the present, rarely planning for the future. This sense of a foreshortened future has a significant impact on the development of life trajectories for the large number of children, adolescents, and young adults who are plagued with this disorder.

While the emotional aspects of PTSD are substantial, the societal, interpersonal, and psychosocial consequences are considerable. People who develop PTSD are more likely to utilize expensive medical services inappropriately, to earn less and divorce more often than the general population, and to become involved with the legal system; they also report greater dissatisfaction with their lives, have more trouble raising their children, and change jobs frequently (Koss, Koss, & Woodruff, 1991; Kulka et al., 1990). Thus exposure to traumatic events and the subsequent development of PTSD constitute a major problem for the public health of this nation and the world. When rates of exposure and PTSD are considered in conjunction with the costs associated with PTSD, efforts to prevent the occurrence of traumatic events and their psychological sequelae become a universal priority. The development of successful treatments for the survivors who develop PTSD also becomes an important public health goal. As with many disorders and condi-

tions, we have accomplished more in terms of developing treatments than we have in prevention. This chapter outlines our achievements to date and the directions for further work.

INCIDENCE, PREVALENCE, AND COURSE

As noted above, when PTSD was initially conceptualized in the DSM-III, both it and the exposure to traumatic events were considered relatively rare conditions. Since the incorporation of PTSD into the diagnostic nomenclature, numerous epidemiological studies have been completed. In addition to examining the prevalence of PTSD, these studies have provided us with information on rates of exposure to traumatic life events, the rates of comorbidity of PTSD with other psychiatric conditions, and the distribution of PTSD among various subgroups of the population (e.g., adults, adolescents, children; males and females; minorities; and particular groups at risk, such as war veterans). They have also helped to establish factors that affect the onset and course of PTSD.

Most studies on the prevalence of traumatic events and PTSD have examined these rates in the United States. Few methodologically sound studies pursuing general population estimates of PTSD have been conducted in other parts of the world. Yet the vast majority of the wars, violence, and natural disasters occurring in the 20th and into the 21st centuries have actually occurred in the developing world. With increasing recognition of the health and economic costs associated with psychological morbidity across the world (Murray & Lopes, 1996), there is a growing acknowledgment of the need for regional and world estimates of psychiatric disorders, including PTSD.

PTSD in the General Population

The first study that attempted to examine the prevalence of PTSD in the general population was the Epidemiologic Catchment Area (ECA) study. Helzer, Robins, and McEvoy (1987) examined data from the St. Louis site only (of the five sites included in the study). As measured by the National Institute of Mental Health's Diagnostic Interview Schedule (DIS), the prevalence of PTSD was observed to be 1% of the general population. Rates of PTSD were higher in women than in men. Scholars criticized this study because of the limited sampling of the population, the manner in which traumatic events were elicited (i.e., asking the participants to categorize the events as traumatic), and the accuracy of the DIS PTSD module (Keane & Penk, 1988). Most leaders in the field felt that the findings were underestimates of PTSD.

Breslau, Davis, Andreski, and Peterson (1991) reported the results of a survey of 1,007 randomly selected members of a health maintenance organization (HMO) in the Detroit, Michigan area. The sample was one of young adults aged 21–30 years. Using an update of the DIS that attempted to address the limitations of the earlier version, they found a prevalence rate for the general population of 9.5%, with 11.3% in women and 5.6% in men. They also found that nearly 40% of the participants had experienced one or more of the traumatic events about which they inquired, a striking finding in itself.

As noted in Chapter 1, perhaps the most complete general population study was that conducted by Kessler and his colleagues (e.g., Kessler et al., 1995), the National Comorbidity Survey (NCS). The investigators interviewed 5,877 nationally representative individuals in the United States from the ages of 15 to 54 years, using an updated version of the DIS, while recording prevalence of different types of traumatic events and their impact. They found

an overall prevalence rate of PTSD of 7.8%, with the rate for women (10.4%) more than twice that for men (5.0%). Estimates for trauma exposure indicated that about 60% of men were exposed to one or more traumatic events, while 51% of women were so exposed.

The NCS also provided information on the types of events most likely to lead to PTSD. Rape and combat were the life events most likely to produce PTSD. The sudden death of a loved one, a very common experience across the sample, was actually the single event that yielded the most cases of PTSD in NCS, even though combat and rape were more likely to result in PTSD from a probability perspective.

There were important demographic differences between those who did and did not develop PTSD. In addition to the large gender difference, Kessler et al. (1995) found a positive association between age and trauma exposure for males and females; as well, there was a slight negative correlation between age and PTSD. These findings indicate, expectedly, that as people age they are more likely to be exposed to traumatic events, but that when they are exposed at an older age there is a trend toward less likelihood of developing PTSD. Kessler et al. also found that marriage seemed to confer some level of protection, as those who were currently married, as opposed to previously married, separated, or widowed, had lower rates of PTSD even when trauma exposure was held constant.

Focusing upon sexual abuse of college-age women, Koss, Gidycz, and Wisniewski (1987) surveyed 3,187 college women about sexual assault and rape. They found that 15.4% of the sample reported that they had been raped, and that another 12.1% reported that someone had attempted to rape them. This study did not collect information about PTSD, but it did set the stage for the National Women's Study, which examined many types of traumatic events in women and corresponding psychological disorders. Kilpatrick, Edmunds, and Seymour (1992) studied 4,008 women, using telephone interviews and the DIS. Thirteen percent of the women of all ages reported a completed rape, 32% of these reported lifetime PTSD. Twelve percent of those raped still had rape-related PTSD at the time of the survey.

Being the target of, or otherwise affected by, a crime is another type of traumatic event that can lead to psychological distress. In a national study of exposure to crime among women in the United States, Resnick, Kilpatrick, Dansky, Saunders, and Best (1993) found that 36% of women reported being directly affected by crime at some point in their lives. Attempted rape or molestation was most frequently reported (14%); death of a friend or family member by homicide was next (13%); experiencing a completed rape was third highest (12.7%); and being physically assaulted was fourth highest (10%). They found an overall PTSD prevalence rate of 12% lifetime and nearly 5% current (within the past 6 months). Resnick et al. found, as have others, that exposure to interpersonal violence increased the likelihood that women developed PTSD. Overall, rates of lifetime (25.8%) and current (8.9%) PTSD were higher for those exposed to crime than for those not exposed (9.4% and 3.4%, respectively).

In a study of adults residing in four Southeastern urban communities, Norris (1992) examined rates of exposure to traumatic events and PTSD. She asked those interviewed in the survey whether they had been exposed to any of eight broad categories of traumatic events. Current rates of PTSD were reported to be 5%, with PTSD most likely to occur following sexual assault (14%), physical assault (13%), and motor vehicle accidents (12%). Clearly, the prevalence of exposure to traumatic events is far more common than anyone anticipated in 1980 when the diagnosis of PTSD was incorporated into the diagnostic nomenclature. Exposure to trauma is common. Even more surprising is that the rate of PTSD in the general population falls only behind the rates of alcoholism, major depression, and social phobia, making it the fourth most common psychiatric condition in the

United States. The high level of disability often associated with PTSD clearly makes it a major public health problem.

PTSD in Combatants

Clearly certain subgroups within our society are at increased risk for being exposed to traumatic life events and for subsequently developing PTSD. The soldiers we send to fight wars and to keep peace are among those most at risk. Orner (1992) reported that in Europe alone there have been over 60 incidents involving military action or terrorism since 1918. The total number of wars across the world in the last half of the 20th century was estimated in the early 1990s at about 150 (Zwi, 1991). Yet few countries have ever tried to assess the psychological toll of war.

The major exception to this was the Vietnam War. The National Vietnam Veterans Readjustment Study (NVVRS; Kulka et al., 1990) was the first time that any government attempted to systematically understand the psychological costs of participating in a war. Employing a two-stage methodology (Dohrenwend & Shrout, 1981), lay interviewers evaluated a representative sample of Vietnam theater veterans (VTVs; n = 1,632), a matched sample of Vietnam era veterans (VEVs; n = 716) and civilian comparison subjects (n = 668). In the second stage of the study, all individuals seen as positive cases in Stage 1 were again interviewed, this time by a clinician using a structured diagnostic instrument (the Structured Clinical Interview for DSM [SCID]; Spitzer, Williams, Gibbon, & First, 1985). A particularly impressive fact about the design of this study was its inclusion of samples of women, African Americans, and Hispanic Americans, which would permit an analysis of the impact of the war on these important subgroups of those who served in Vietnam.

The findings on exposure to traumatic events were rather striking. Sixty-four percent of the VTVs were exposed to one or more traumatic events in their lives in contrast to 48% among the VEVs and 45% among the civilians. Clearly, the exposure rates across all groups were high. To make the diagnosis of PTSD, the NVVRS investigators relied upon a triangulation approach that utilized information from the diagnostic interviews as well as several self-report questionnaires that were administered (e.g., the Mississippi Scale; Keane, Caddell, & Taylor, 1988). Findings indicated that more than 15% of male VTVs met criteria for current PTSD and 30% met criteria for lifetime PTSD. Among the female VTVS, 9% met criteria for current PTSD and 27% met criteria for lifetime PTSD. In all cases, these prevalence rates were 5–10 times higher than those found for the VEVs and the civilians. These findings indicated that there were approximately 479,000 cases of current PTSD and nearly 1 million cases of lifetime PTSD in America stemming from the Vietnam War alone.

The NVVRS also reported findings for African American and Hispanic groups that served in Vietnam, as noted above. Of the approximately 3.14 million Americans who served in Vietnam nearly 170,000 were Hispanic Americans and 350,000 were African Americans. There were different rates for current PTSD among the various ethnic and racial groups: For the European American/other group the prevalence of current PTSD was 13.7%, while for the African Americans it was 20.6% and the Hispanic Americans it was 27.9%. The differences were largely due to higher levels of combat exposure among the minorities. Gender differences were also apparent in the NVVRS: Female VTVs had lower rates of PTSD than did the males. However, much of this difference must be attributed to the different roles that women had in the military at that time (primarily nursing and clerical), the different types of stressors to which they were exposed, and the higher educational levels among the women.

The NVVRS was a rare study in that it had an instant effect on public policy toward war veterans in America. Based on the findings, Congress allocated increased resources to address the psychological and social effects of war on veterans. These programs continue today and serve veterans of all wars, conflicts, and peacekeeping missions. In recognition of the prevalence of sexual assault among women veterans, programs have expanded to address the needs of sexual assault survivors as well.

There has been considerable debate about the impact of the Persian Gulf War on the soldiers sent to free Kuwait. Sutker, Uddo, Brailey, and Allain (1993) examined a convenience sample of 215 troops sent to the Persian Gulf. They found a prevalence rate of 16–19% for PTSD. Wolfe, Brown, and Kelley (1993) conducted a longitudinal study of 2,344 Persian Gulf veterans and found prevalence rates of PTSD based upon the Mississippi Scale (Keane et al., 1988) of 4% for men and 9% for women. Perconte et al. (1993) found a prevalence rate of 16% among 439 reservists sent to the Persian Gulf, whereas a prevalence of less than 4% was found for reservists deployed elsewhere or who remained at home. Clearly, the exposure to combat and war-related traumatic events yielded discernible levels of PTSD among various Persian Gulf veteran cohorts. Still under discussion is the extent to which the military in the Persian Gulf was exposed to toxicants (e.g., depleted uranium, burning oil, pesticides), which might be responsible for the increased levels of disorder and disability observed following that war. It is likely that these factors all interacted with the stress of deployment, combat, and reentry to yield the diverse psychological and health problems in this most recent cohort of American veterans.

Since the end of the Cold War, the United States and other leading nations have intervened, often with the United Nations, in efforts to maintain peace in many areas of the world. Peacekeeping has its own set of associated stressors, and increasingly these are being recognized as leading to PTSD. For example, peacekeeping forces are often instructed not to fire unless others fire upon them; they are also asked to patrol areas that are often under tenuous control. It is reasonable, then, to ask whether peacekeeping duties can lead to PTSD. Litz, Orsillo, Friedman, Ehlich, and Batres (1997) examined a sample of 3,461 active-duty military troops who served in Somalia, a particularly gruesome peacekeeping assignment for U.S. soldiers. Studied shortly after their return to the United States, these soldiers manifested a prevalence rate of PTSD of 8%—a rate that didn't differ for men and women. Clearly, those who serve as peacekeepers across the world are at risk for developing PTSD, even though the actions are not seen as traditional combat.

PTSD in Disaster Contexts

There is a burgeoning literature supporting the idea that a wide range of natural disasters and accidents can lead to the development of PTSD. Green and her colleagues first studied the psychological impact of the collapse of the Buffalo Creek Dam in West Virginia in the late 1970s. Initially they found that 44% of the survivors reached criteria for PTSD. When the survivors were studied some 14 years later, 28% of the sample still met criteria for PTSD, indicating that the effects of this type of disaster are long-lasting (Green, Lindy, Grace, et al., 1990; Green, Grace, Lindy, et al., 1990).

Similarly, McFarlane (1989) studied the effects of bush fires in Australia. These fires often burn for days and weeks, leaving disaster in their wake and challenging even the best firefighting teams to predict or to control them. Death, injury, and destruction are commonplace. Examining 315 firefighters, McFarlane found prevalence rates of PTSD of 32%, 27%, and 30% respectively at 4, 11, and 29 months after the disaster. Again, therefore, high

rates of PTSD followed a disaster—rates that were sustained for a substantial period of time following the exposure.

These are just a few of the many different types of disasters that have been studied successfully over the past 20 years. These elevated rates and figures are not unusual; rather they are more typical than not. Studies of Three Mile Island (Bromet et al., 1982), Chernobyl (Havenaar et al., 1997), and the Lockerbie air disaster (Brooks & McKinlay, 1992) all indicate pervasive traumatic reactions in survivors, regardless of age, gender, or socioeconomic status. The maturing epidemiological literature on the effects of trauma exposure and PTSD has yielded considerable evidence to suggest that these events themselves contribute substantially to the development of PTSD and related disability and dysfunction.

PTSD in Children

PTSD is also common in children and adolescents. Kilpatrick and Saunders (1999) conducted a nationally representative survey of adolescents that included diagnostic interviews. They found that in adolescents under the age of 18, the current prevalence rate of PTSD was 5% of the U.S. population. Again, prevalence was higher among females than among males. The events most likely to yield PTSD were abuse and violence, often at the hands of family members.

Although there is still no nationwide epidemiological study of psychiatric conditions in children, there are excellent studies of the exposure of children to violence. Bell and Jenkins (1993) studied 536 African American elementary students (grades 2, 4, 6, and 8) at three different schools in urban Chicago. Strikingly, 26% of the students had seen someone being shot, and 30% had observed someone being stabbed. Moreover, 75% of the students stated that they had been exposed to at least one violent crime. Similar reports of exposure to violence in children abound in the literature (e.g., Burton, Foy, Bwanausi, Johnson, & Moore, 1994; Singer, Anglin, Song, & Lunghofer, 1995).

There are many studies examining rates of stress and PTSD among specific subgroups of children and adolescents. For example, Saigh (1988) examined civilian adolescents during various periods of war stress in Lebanon. He found that as time intervals reflecting high levels of combat alternated with periods of peace, these children systematically reported elevations in anxiety and depression and low levels of assertiveness. These changes reflected functional impairments as well, with the children having increasing difficulties at home and at school as they experienced stress secondary to the war.

In their evaluations of the survivors of the Buffalo Creek Dam collapse, Green and her colleagues found that among the 179 children they studied, some 37% had "probable" PTSD. Following Hurricane Andrew, a natural disaster, La Greca, Silverman, Vernberg, and Prinstein (1996) found a 30% prevalence rate of PTSD at 3 months, 18% at 7 months, and 13% at the 10-month follow-up among school-age children. These studies of selected high-risk samples of the population indicate that when children and adolescents are exposed to the death and destruction accompanying technological and natural disasters, they experience high rates of PTSD.

The Armenian earthquake of 1988 was one of the most dramatic of the natural disasters studied to date, in part because of the high levels of destruction near the earthquake's epicenter. Goenjian et al. (1995) found a PTSD prevalence among the children closest to the epicenter (mean age = 13 years) of 95%; among those children at a moderate distance from the epicenter, there was a PTSD prevalence of 71%; and for those furthest from the destruction, there was a PTSD prevalence of 26%. The linear relationship between intensity of the experience and the development of PTSD demonstrates the

powerful effect of the traumatic event per se in the development of this psychological condition.

Finally, Garrison, Weinrich, Hardin, Weinrich, and Wang (1993) found a prevalence rate of 5% current PTSD in over 1,200 students who had been exposed to a disastrous hurricane in South Carolina. In all of these studies on disasters, the selection of participants for inclusion in the sample was not representative of the general population in the areas studied. For this reason, conclusions must be drawn tentatively; yet these studies provide an excellent base from which additional studies might be conducted following major life stressors. Clearly, the evidence suggests that either technological or natural disasters can yield high rates of PTSD, and that (as in adults), the more directly children or adolescents are affected, the more likely they are to develop PTSD.

PTSD Following Childhood Sexual Abuse

Childhood sexual abuse is one crime that has gotten increasing attention in the scientific literature since the publication of the classic book *Father–Daughter Incest*, by psychiatrist Judith Herman (1981). Growing empirical data suggest that the true prevalence of childhood sexual assault and the resulting psychological impact of this abuse are both greater than initially thought (Finkelhor & Dziuba-Leatherman, 1994). Past studies were hampered in part by the reporting requirements of governmental agencies and in part by the reluctance of family members to come forward when evidence of abuse (especially intrafamilial abuse) is uncovered (Freyd, 1994). As well, there is still no nationwide study that has attempted to tackle the many technical, methodological, political, and ethical issues involved in investigating sexual abuse in children and its relationship to psychiatric disorder (e.g., PTSD). Still, a number of studies of convenience samples indicate the difficulties experienced by some children who are identified as having been sexually abused.

McLeer, Deblinger, Atkins, Foa, and Ralphe (1988) studied 32 patients at a university clinic for sexually abused children. Using a locally constructed interview, they found that 48% of the participants met DSM-III criteria for PTSD. Similarly, McLeer, Deblinger, Henry, and Orvaschel (1992) examined 92 children who had been sexually abused and reported that 44% of the children reached criteria for PTSD, with those abused by their parent or a trusted adult more likely to have PTSD than those abused by a stranger or older youth.

In New Zealand, Merry and Andrews (1994) studied 66 children 3 to 6 months following the index sexual abuse event. Some 64% of these children met criteria for a psychiatric condition, and 18% of the sample met criteria for PTSD. These findings support the concept that traumatic events can precipitate PTSD as well as other psychiatric conditions. Interestingly, in Canada, Wolfe, Sas, and Wekerle (1994) used a locally constructed PTSD scale to examine 90 children with court-documented childhood sexual abuse. In their sample, Wolfe et al. (1994) found that 49% met DSM criteria for PTSD—a considerably higher rate than in the Merry and Andrews (1994) study.

Methodological differences in the studies discussed above limit the extent of our conclusions about the prevalence rate of PTSD in the general population of children and adolescents. Particularly problematic is the absence of measures that are widely accepted for measuring PTSD in children and adolescents. Although there have been several excellent attempts to address this limitation (Fredrick, Pynoos, & Nader, 1992; Nader et al., 1998; Saigh, 1989), to date no single instrument has proved itself to be the assessment device of choice for children and adolescents. This has clearly limited the extent to which sound epidemiological studies of the prevalence of PTSD in children can be conducted.

A MODEL OF THE ETIOLOGY OF PTSD

As noted earlier, we do not yet know the "cause" of PTSD, but identification of the precipitating event or proximal cause is relatively simple. In this sense, the etiology of PTSD seems much more straightforward, based on the theoretical descriptions of anxiety and fear described in Chapters 7 and 8. Unlike specific phobia, where true alarms, false alarms, or (less often) simple transmission of information may develop into a phobic reaction, PTSD seems to emerge from one special chain of events. Intense basic emotions, such as true alarms (but also including rage or distress, resulting from the overwhelming effects of traumatic life events), lead to learned alarms. Learned alarms occur during exposure to situations that symbolize or resemble an aspect of the traumatic event. This is one of the defining features of PTSD in DSM-IV (American Psychiatric Association, 1994).

Among occasions that symbolize aspects of the traumatic event are anniversaries of the trauma. As in any phobic reaction, the development of learned alarms results in persistent avoidance of stimuli associated with the trauma, which is another defining feature of PTSD in DSM-IV. Other stimuli associated with the trauma include thoughts or feelings, as well as memories of the event.

Wirtz and Harrell (1987) provide support for the process of classical conditioning (learned alarms) within PTSD. They observed that survivors of physical assault were less distressed 6 months after the assault if they had experienced exposure to situations or stimuli that were part of (or resembled) the context of the original assault without experiencing another assault. Survivors who had not had the advantage of this exposure, on the other hand, maintained a high level of distress in the 6-month interim. This is what one would expect in classical conditioning. Others have noted the seeming importance of conditioning in the development of PTSD (Keane, Fairbank, Caddell, Zimering, & Bender, 1985; Orr et al., 2000), whether the trauma is combat-related, involves a physical assault such as sexual assault (e.g., Holmes & St. Lawrence, 1983; Kilpatrick, Veronen, & Best, 1985; Orr et al., 1998), or is the consequence of terrorism (Shalev et al., 2000).

This suggests an important similarity to panic disorder that has been alluded to above. In PTSD, the experience of affect itself is avoided to some extent; this is characterized clinically as a numbing of general responsiveness. This seems to be similar to the tendency of patients with panic disorder to avoid feelings such as those occasioned by movies, whether they be frightening, sad, or exciting. According to the conceptualizations presented above, fear is associated with interoceptive cues signaling the possible occurrence of another false alarm (learned alarm). The numbing of general responsiveness in PTSD would seem to represent avoidance of aversive emotional reactions or alarms (Jones & Barlow, 1990; Litz, 1992; Litz et al., 2000). Whether the sensitivity to or avoidance of interoceptive cues in panic and the numbing of general responsiveness in PTSD are identical psychopathological responses remains to be demonstrated.

In any case, the experience of alarm or other intense emotions is not sufficient in and of itself for the development of PTSD. Much as in other disorders, one must develop anxiety or the sense that these events, including one's own emotional reactions to them, are proceeding in an unpredictable, uncontrollable manner. When negative affect (including a sense of uncontrollability) develops, one enters the vicious cycle of anxious apprehension described in Chapter 3, and PTSD emerges.

This implies that a psychological and biological vulnerability to develop the disorder exists, as outlined in Chapter 8. There, it is also noted that anxiety is always moderated to some extent by variables such as the presence of adequate coping skills and social support. In PTSD (as indicated in the work of the Kings; see "Modeling the Prediction of PTSD,"

below), evidence already exists that these moderating variables play a role in determining whether the disorder develops or not. Therefore, these factors are represented explicitly in the model presented in this chapter, although it is assumed that these factors moderate the occurrence of other anxiety disorders to an equal extent. A model of the etiology of PTSD is presented in Figure 12.1. An elaboration of the evidence supporting various aspects of the model is presented below.

RISK FACTORS

Interest in risk factors has grown considerably in recent years. In their comprehensive and illuminating review of risk factors entitled "Coming to Terms with the Terms of Risk," Kraemer et al. (1997) define the various forms of risk factor analysis that accompany epidemiological studies. In particular, these authors emphasize that risk factors are true correlates of a particular disorder. Risk factors differ from simple correlates, in that they are found to precede the onset of the disorder itself. They are then considered to be candidates for "causal risk factors," or ones that contribute to the etiology of the disorder.

Interest in risk factor analysis is important, because the identification of risk factors may assist us in the development of preventative approaches for people who are at highest risk for developing the disorder. These interventions, in the case of PTSD, may precede the occurrence of a traumatic event or may help us to identify those people at greatest risk for developing PTSD when exposed to massive traumatic events such as country- or region-wide technological disasters (e.g., the Chernobyl nuclear disaster). The study of risk factors, then, takes many different forms, some of which are examined below for their relevance to helping us better understand the etiology of PTSD.

Family Studies

What factors might account for the development of PTSD in some individuals following exposure to a major life stressor, but not in others undergoing seemingly the same stressor?

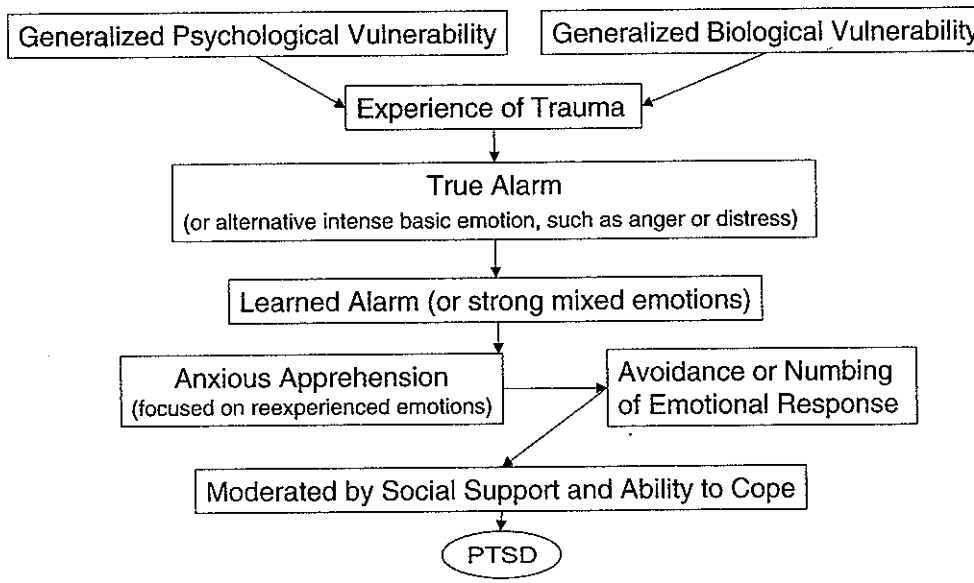

FIGURE 12.1. A model of the etiology of PTSD.

As reviewed above, a generalized biological vulnerability consisting of constitutional and hereditary variables is a major focus. Other contributing variables include the extent of exposure, the number of exposures to the same or similar traumatic events, the coping strategies of the individual, the availability of social support, and a generalized psychological vulnerability to developing anxiety/negative affect.

Examination of the evidence surrounding the constitutional/hereditary contributions to PTSD yields little systematic evidence to date that bears on this issue. Discriminating the predisposition for exposure to traumatic events from the predisposition to developing PTSD also represents a methodological and interpretive challenge. For example, people who are risk takers (e.g., skydivers) may actually be more likely to be exposed to traumatic events. Will they also be more or less likely to develop PTSD once exposed? Do the factors that predispose them to be risk takers function to prevent them from developing PTSD? These are questions of profound importance to accurately identifying those variables that contribute to the development of PTSD.

What has the literature told us about candidate risk factors for PTSD? Curran and Mallinson (1940) compared the family histories of 100 British soldiers returning from World War II with psychoneurosis to those of 50 surgical controls. Forty-five percent of the psychiatric casualties had a family member with a past psychiatric hospitalization, functional impairment, or a suicide attempt—a percentage considerably higher than that in the comparison group.

Cohen, White, and Johnson (1948) compared the family histories of 144 World War II service personnel with neurasthenia to those of 105 healthy comparisons, and 48 veterans and medical patients. The authors noted particularly high rates of neurasthenia in the mothers of the index group (58%), but also in the fathers (19%) and in the siblings (13%). The authors reported that no such histories were found in the comparison groups. Importantly, this study also interviewed a small subgroup of family members in the index group ($n = 15$) and confirmed the reports of the patients themselves. This was among the earliest efforts to incorporate family interviews into studies of family history of psychopathology.

Davidson, Swartz, Storck, Krishnan, and Hammett (1985) conducted the only family study to date of PTSD using contemporary methods of interviewing and diagnosis. Selecting patients from World War II with diagnoses of PTSD, they found familial rates of disorder in 66% of the participants. Of course, these patients were seeking help and may not truly represent PTSD in the general population; nonetheless, these data provide some preliminary support for the idea that there is a hereditary contribution to the development of PTSD. Similarly, in his examination of survivors of disastrous bush fires in Australia, McFarlane (1988) reported that 55% of those with PTSD had relatives with a psychiatric condition, whereas only 20% of those who did not develop PTSD reported such a family history.

In addition, two epidemiological studies indicated a possible family contribution to the development of PTSD. Davidson, Hughes, Blazer, and George (1991) examined the ECA data from the North Carolina site and observed that people with PTSD were three times more likely to report family psychiatric disorders than were comparison participants. In the Detroit HMO study, Breslau et al. (1991) observed elevations in reported family rates of several anxiety, mood, and psychotic disorders among those with PTSD. These epidemiological studies are all subject to problems with bias in retrospective recall. Although they do provide some important preliminary evidence, there is a need for future studies in this area to use contemporary methods of sampling, diagnosis, and family interviews, in order for more firm conclusions to be drawn on the role of constitutional/hereditary factors in the development of PTSD.

Twin studies have also provided some evidence supporting a constitutional basis for PTSD. True et al. (1993), using the Vietnam Veteran Twin Registry, found heritability factors for exposure to combat as well as to several of the PTSD symptoms. Yet this study used only questionnaire data in the analysis, and so strong conclusions must be deferred. Pitman and his colleagues (2000), using this same twin registry, attempted to elucidate the parameters that might underlie PTSD. Using a wide variety of laboratory tasks (e.g., psychophysiological reactivity, evoked related potentials) and standardized diagnostic tools, they found little evidence for an inherited component for PTSD.

In summary, there is preliminary evidence suggesting that there may be a constitutional/hereditary component for PTSD. Yet most studies conducted to date have methodological and procedural limitations that compromise our capacity to draw strong inferences. Pitman et al. (2000) have presented only the preliminary analyses of their excellent twin study, and these did not seem to identify key constitutional variables. Additional studies will be required in order to identify those biological and physiological variables that interact with traumatic events to produce PTSD. At best, we can conclude that the presence of a family history of psychopathology increases the likelihood that one will develop PTSD once exposed to a traumatic event. Although the precise nature of this relationship remains to be further explored, it is likely that a generalized neurobiological tendency to react in an exaggerated manner to stress—described in Chapters 6 and 8 as associated with the development of all anxiety disorders and depression—also describes the generalized biological vulnerability to develop PTSD.

Demographic Risk Factors

Epidemiological studies of trauma exposure and PTSD provide one important way in which we can determine who develops PTSD following certain traumatic events. The NCS (Kessler et al., 1995), as noted earlier, included a representative sample of males and females between the ages of 15 and 54 from the 48 contiguous states. Information about PTSD was collected in face-to-face interviews on a subset of participants (5,877 of the 8,098 total). Consistent with prior research on women (Kilpatrick & Resnick, 1992), PTSD was found to vary as a function of the type of traumatic events experienced; rape, childhood physical abuse, and childhood neglect were the most likely events to lead to PTSD in this national sample. Each of these three types of events was also more likely to occur in women. Although men were more likely to report a traumatic event than women, women were more likely to report a traumatic event that was strongly linked to developing PTSD.

Breslau, Chilcoat, Kessler, Peterson, and Lucia (1999), in the Detroit HMO trauma study, examined the link between gender and PTSD in an effort to determine whether the higher prevalence of PTSD consistently observed among women is simply a function of the types of events to which they are differentially exposed. They found that the risk of developing PTSD among women was approximately twice that of men even when types of traumatic events were taken into account. Findings from the NCS supported these conclusions as well. The precise nature of the mechanism involved in the sex difference in PTSD has not yet been explained, but it is a consistent finding across most anxiety disorders, and is a question of such high importance that it will undoubtedly receive considerable attention in the future. Hypothetical explanations for a differential generalized psychological vulnerability based on gender are reviewed in Chapter 8.

In addition to gender, age has been examined as a risk factor for exposure to traumatic events and to developing PTSD. Among women, the NCS found no relationship between age and PTSD, and only a small relationship between age and exposure to trau-

matic events. In contrast, among men there was a strong positive correlation between age and PTSD, and this was due to increasing exposure to traumatic events over the lifespan.

The findings regarding race as a risk factor are somewhat complicated. To draw again upon the NCS data, African Americans and other minorities (Asian Americans and Native Americans) reported fewer exposures to traumatic events than did European Americans and Hispanic Americans. Yet these same groups (i.e., African Americans and other minorities) reported higher rates of PTSD following exposure. In the NVVRS, the study of the effects of the Vietnam War, prevalence of PTSD also varied by racial/ethnic subgroups. The prevalence rate among Hispanic Americans was about 28%; for African Americans, it was 21%; and for European Americans and others, it was 14%. These findings were largely, but not entirely, due to differences in rates of combat exposure. Other factors, especially among the Hispanic veterans, also contributed to the development of PTSD. These factors were never conclusively articulated and remain important to investigate. In sum, the role of race in the development of PTSD must take into account different levels of exposure to traumatic events. Yet even when this is done, there are still factors contributing to the differential rates of PTSD and trauma exposure that are observed in epidemiological studies. The differences found across race and gender are important for future research to address. They may provide important keys to solving the complex puzzle that PTSD presents.

There are other factors as well that relate to the development of PTSD. Several studies (the NCS, the NVVRS, and the Detroit HMO study) identify the prior existence of a psychiatric condition as a risk factor for the development of PTSD, once an individual is exposed to a traumatic event. In addition, the presence of an addictive disorder and/or of conduct disorder may well *lead* to exposure to traumatic events (Breslau et al., 1991; Kessler et al., 1995). This complex interaction is fundamental to our understanding of the effects of traumatic events and PTSD. Some psychological conditions, such as panic, depression, and social anxiety, can lead to the use of addictive agents (e.g., alcohol, heroin, cocaine); these can in turn create a lifestyle that results in exposure to assaultive violence, which then leads to the development of PTSD (Keane & Wolfe, 1990). Alternatively, a sexual assault can lead to the development of panic attacks, social anxiety, depression, or PTSD, which then leads to the use of addictive substances. Both patterns are commonly observed in epidemiological studies and provide important information for clinicians treating patients. The relationship between anxiety and substance use disorders is reviewed in some detail in Chapter 1. Careful assessment of the precipitating variables that contribute to a particular psychological condition can provide meaningful information about which condition to treat first in patients with multiple comorbid psychological disorders (e.g., Najavits, 2001).

Modeling the Prediction of PTSD

To understand more fully the factors associated with who does and who doesn't develop PTSD following a traumatic event, it would be necessary to assign individuals randomly to certain types of traumatic events and then follow them over time to record the effects on their lives. Such a strategy would allow us to disentangle some of the puzzles presented by epidemiological risk factor analyses (e.g., the finding that exposure to traumatic events does not appear to be random). Yet ethical standards do not permit research of this type. Structural equation modeling (SEM) is one statistical strategy that attempts to understand the direction of effects when many factors are involved in predicting an outcome.

SEM has been applied to the understanding of risk factors in PTSD primarily in war veterans. Psychologists Dan and Lynda King and their colleagues have applied SEM to a wide variety of theoretically driven variables in an effort to predict which men and women developed PTSD following the Vietnam War. Using the NVVRS data set, they created four war zone stressor variables: traditional combat, atrocities/abusive violence, perceived threat, and the malevolent environment of the war zone (King, King, Gudanowski & Vreven, 1995). Atrocities/abusive violence, perceived threat, and malevolent environment all had direct effects on PTSD outcome, with malevolent environment exerting the largest effect. Traditional combat had an indirect effect, influencing the development of PTSD primarily through the perceived threats that the individuals reported.

Next, King, King, Foy, and Gudanowski (1996) examined prewar factors, demographic variables, and war zone stressor variables and their relationship to PTSD. The prewar variables consisted of measures of family environment, childhood antisocial behavior, maturity at entry to Vietnam, and prior trauma exposure. For both men and women (the investigators derived separate equations for each), they found that war zone stressors remained important contributors to PTSD, but that additional variance was attributable to the prewar factors for men and women. The effect of prewar variables was greater for men than it was for women. For men, a prior history of trauma exposure and age at the time of trauma were important factors; for women, only the history of a prior traumatic event contributed to the development of PTSD. These findings accentuated the importance of family instability and of a previous trauma history contributing to a generalized psychological vulnerability (see Chapter 8) as determinants of who develops PTSD.

King, King, Fairbank, Keane, and Adams (1998) then used SEM to examine resilience and recovery factors with the NVVRS data set. They identified hardiness, structural social support, functional social support, and recent stressful life events as possible factors that would explain who developed chronic PTSD. When combined with the war zone stressor variables, they learned that the variables promoting resilience differed for men and women. For men, hardiness, structural support, functional support, and stressful life events all had direct effects on PTSD development. For women, only hardiness, functional support, and stressful life events did. The investigators concluded that there are complex patterns of direct and indirect effects of the wide range of variables they incorporated into their models, and that these patterns differed for men and women.

In the final report of their sequence of studies, King, King, Foy, Keane, and Fairbank (1999) aggregated the pretrauma risk factors, the four war zone stressors, and the posttrauma resilience and recovery variables to understand more comprehensively how these variables interact to lead to the development of PTSD. For female VTVs, these three categories of variables predicted 72% of the variability in PTSD as an outcome. Prewar trauma exposure, exposure to abusive violence and life threat during the war, and postwar additional life stressors and functional social support were the most significant factors in determining who among the women who served developed PTSD. Among the male VTVs, these researchers were able to account for 70% of the variance in PTSD as an outcome. The key variables among men were the same as for women, plus younger age at the time of service in the war, the malevolence of the war zone environment, and structural social support (i.e., number and types of supports). Thus, using only measures of environmental and psychological constructs, these researchers were able to identify important risk factors related to the development of PTSD. Future studies that would include constitutional, physiological, and measures of hereditary factors would improve the level of precision possible in prediction. Even still, the levels of precision obtained without the biological level of analysis are impressive indeed.

ASSESSMENT AND DIAGNOSIS

Since its inclusion in the DSM-III in 1980, there has been excellent progress in the psychological assessment of PTSD. Consisting of a wide array of symptoms, PTSD presents a significant challenge to those involved in the development of assessment instruments. Yet it is clear that this challenge has been successfully met with an assortment of high-quality diagnostic interviews and psychological tests (Keane, Weathers, & Foa, 2000; Weathers, Keane, & Davidson, 2001). The focus of this part of the chapter is on the recent progress attained in the assessment of trauma and PTSD in adults.

An Overview of Assessment

Increasingly, clinicians are recognizing that many patients in their clinical practice have experienced traumatic events. As a result, the psychological care of large numbers of patients in hospitals, clinics, and practices is complicated by the presence of PTSD. Accordingly, there has been growing interest among clinicians in the proper assessment and evaluation of patients with PTSD.

Clearly, PTSD is assessed for many different purposes, and the goals of a particular assessment can determine the approach selected by the professional. A common purpose for assessment is to screen large groups of individuals to determine the extent to which some proportion of individuals have been exposed to traumatic events and have symptoms associated with PTSD. A second purpose for assessment, and perhaps the most common, is the completion of a diagnostic workup that includes a differential diagnosis and treatment planning. Clinicians may also be involved in forensic evaluations, where diagnostic accuracy is of utmost importance. Researchers may be interested in the frequency of occurrence of PTSD and the risk factors and complications associated with it (as in epidemiological studies). Moreover, researchers may be interested in high levels of diagnostic accuracy when studying biological and psychological parameters of the disorder, as in case–control designed studies. Each clinical and research situation requires a different solution, depending upon the assessment goals of the professional.

All measures of a psychological disorder are imperfect (Gerardi, Keane, & Penk, 1989). Two measures of the error contained within a test are the concepts of "false positives" and "false negatives." A false positive occurs when a patient falls above the cutoff, but is not a true case. A false negative occurs when a patient falls below the given cutoff, yet is in fact a true case. Diagnostic utility is often described in terms of a test's "sensitivity" and "specificity." These are measures of a test's performance that take into account errors made in prediction. Sensitivity is the measure of a test's "true-positive" rate, or the probability that those with the disorder will score above a given cutoff score. Specificity is the "true-negative" rate of a test, or the probability that those without the disorder will score below the cutoff for the test. Sensitivity is low if the test yields too many false negatives, whereas specificity is low if the test yields too many false positives.

Selection of tests and diagnostic instruments should include an examination by the clinician of relevant data on their psychometric properties. Inspecting rates of false positives, false negatives, sensitivity, and specificity can also inform the clinician of how an instrument performs. Conclusions drawn in clinical assessment are most accurate if they take into account these limitations.

Efforts to diagnose and assess patients for the presence of PTSD can include a range of different methods. These include semistructured diagnostic interviews for PTSD and related comorbidity, psychological tests and questionnaires, psychophysiological measures,

medical records, and the use of multiple informants regarding the patient's behavior and experiences. This approach has been referred to as a multimethod approach to the assessment of PTSD (Keane, Fairbank, Caddell, Zimering, & Bender, 1985).

Semistructured Diagnostic Interviews

In the practice of clinical research, it is standard to employ a semistructured diagnostic interview to insure that all PTSD symptomatology is reviewed in detail. Diagnostic interviews combine the virtues of defining precisely how a diagnosis was made with those of using interviews that have known psychometric properties (i.e., reliability and validity). The use of structured diagnostic interviews in the clinical setting is less common, with perhaps the single exception of clinical forensic practice, where it is strongly encouraged (Keane, 1995). Nonetheless, the use of diagnostic interviews in clinical settings may well improve diagnostic accuracy and improve treatment planning (Litz & Weathers, 1994). The use of broad-based diagnostic interviews that cover the range of high-frequency diagnoses will assist the clinician by providing an evaluation of not only the target disorder, but also the extent of clinical comorbidity that is present (Keane & Wolfe, 1990; Weiss, 1997). Some of the available diagnostic interviews and their psychometric properties are presented below.

Clinician-Administered PTSD Scale

Developed by the National Center for PTSD in Boston, the Clinician-Administered PTSD Scale (CAPS) was designed for use by trained, experienced clinicians (Blake et al., 1990). Consisting of 30 items, the CAPS assesses all 17 symptoms of PTSD, as well as a range of the frequently observed associated features. Also contained in the CAPS are ratings for social and occupational functioning and an assessment of the validity of the responses by the patient. Like several other interviews (see below), the CAPS provides both dichotomous and continuous scores. Unique features of the CAPS are that it contains separate ratings for frequency and intensity of each symptom, and that it possesses behaviorally anchored probe questions and scale values. Interviewers are trained to ask their own follow-up questions and to use their clinical judgment in arriving at the best ratings.

If administered completely (i.e., all questions regarding associated features, functional impairments, validity ratings), the CAPS takes approximately an hour to complete. If only the diagnostic symptoms are assessed, the time for administration is cut in half.

Psychometric data on the performance of the CAPS demonstrate unusual strength in identifying cases and noncases of PTSD. Across three clinicians and 60 separate male veteran subjects, Weathers et al. (1992) found test–retest correlations between .90 and .98. Internal consistency was equally impressive, with alpha at .94 across all three primary symptom clusters. Correlations with other established measures of PTSD yielded strong evidence for the construct validity of the CAPS. The correlation of the CAPS with the Mississippi Scale for Combat-Related PTSD was .91, with the Keane PTSD Scale of the Minnesota Multiphasic Personality Inventory—2 (MMPI-2) was .77, and with the SCID (see below) PTSD symptom score was .89. Correlations with a measure of antisocial personality disorder were low, as was predicted by the multitrait–multimethod study design.

When the CAPS was used as a continuous measure, it was found to have 84% sensitivity, 95% specificity, 89% efficiency, and a kappa of .78 against the SCID. When the CAPS was used as a diagnostic measure, a kappa of .72 was found as compared with the SCID diagnosis. Whether it is used as a diagnostic or a continuous measure, these findings

establish the CAPS as a sound measure of PTSD with excellent psychometric properties. Replications of these findings with male and female survivors of motor vehicle accidents (Blanchard et al., 1995) and patients with serious mental illnesses of both genders (Mueser et al., 1999) indicated the generalizability of these results across populations, races, and genders. A recent publication has carefully explicated nine different scoring algorithms for the CAPS and their implications for diagnostic accuracy, reliability, and validity coefficients (Weathers, Ruscio, & Keane, 1999).

Structured Clinical Interview for DSM

The SCID (Spitzer, Williams, Gibbons, & First, 1994) is the most widely used interview to assess Axis I and Axis II psychiatric disorders. It consists of separate modules for the most common of the diagnostic categories. Although the administration of the full SCID can be time-consuming, it does provide information across a broad range of clinical conditions. In many clinical settings, the SCID is used to systematically assess only those conditions that are most frequently encountered. This is economical in terms of time and still provides an examination across key conditions. In working within the context of a trauma clinic, it is recommended that the anxiety disorder, mood disorder, and substance use disorder modules and the psychotic screen be employed. This provides a fairly comprehensive examination of those conditions that are frequently comorbid with PTSD, and it provides a systematic way in which to insure that a patient does not endorse signs of psychoses (conditions that would require a different initial set of clinical interventions).

The PTSD module of the SCID appears to be both clinically sensitive and reliable. Keane et al. (1998) examined the interrater reliability of the SCID by asking a second interviewer to listen to audiotapes of an initial interview. They found a kappa of .68 and agreement across lifetime, current, and "never" PTSD of 78%. Similarly, in a sample of patients who were reinterviewed within a week by a different clinician, they found a kappa of .66 and diagnostic agreement of 78%.

The SCID's primary limitation is that it permits only a dichotomous rating of a symptom (present or absent), placing clinicians in a forced-choice situation. Most clinicians agree that the psychological symptoms occur in a dimensional rather than a dichotomous fashion, and so the SCID seems limited by the use of the present–absent scoring algorithm. Several options have evolved in the field as a result of this limitation.

Anxiety Disorders Interview Schedule

The original Anxiety Disorders Interview Schedule (ADIS) was developed almost 20 years ago (Di Nardo, O'Brien, Barlow, Waddell, & Blanchard, 1983). It was revised for DSM-III-R (ADIS-R; Di Nardo & Barlow, 1988), and revised again for DSM-IV (ADIS-IV: Lifetime Version; Di Nardo, Brown, & Barlow, 1994; see Chapter 9). The ADIS is a structured diagnostic interview that focuses primarily on the anxiety and mood disorders. The ADIS uses a Likert-type scaling procedure for symptoms and is thus capable of being analyzed in multiple ways to determine the extent to which a symptom is present or absent. Psychometric properties of the ADIS PTSD module have been assessed in two separate studies, and there were mixed results. In the first study a small group of combat veterans was assessed by two independent interviewers. Blanchard, Gerardi, Kolb, and Barlow (1986) found excellent sensitivity (1.0) and specificity (.91) for the original ADIS. In a community-based study of the ADIS-R, the results were slightly less impressive, and the hit rates were less stable (Di Nardo, Moras, Barlow, Rapee, & Brown, 1993).

PTSD Interview

The PTSD Interview (Watson, Juba, Manifold, Kucala, & Anderson, 1991) yields both dichotomous and continuous scores. The authors report strong test–retest reliability (.95) and internal consistency (alpha = .92), as well as strong sensitivity (.89), specificity (.94), and kappa (.82) when this interview is compared with the DIS (Robins, Helzer, Croughan, & Ratcliff, 1981).

The PTSD Interview appears to have excellent psychometric properties, but differs in administrative format from most other diagnostic clinical interviews. With the PTSD Interview, the clinician provides the patient with a copy of the scale to read along with the interviewer. From this copy of the scale, the patient is asked to give to the clinician his or her rating on the Likert scale for each of the symptoms. This format has much in common with self-report questionnaires, but it deviates from the other diagnostic scales in that it does not allow clinicians to make ratings of their own and utilize their expertise and experience.

Structured Interview for PTSD

The Structured Interview for PTSD (SI-PTSD) was developed by Davidson, Smith, and Kudler (1989). Like the CAPS and the PTSD Interview, it yields both dichotomous (i.e., diagnostic) and continuous measures of PTSD symptoms. As a result, it appears to be a useful instrument for diagnosing PTSD and measuring symptom severity. Symptoms are rated by the clinician on 5-point Likert scales, and the focus for the clinician is on symptom severity. The SI-PTSD possesses initial probe questions and provides helpful follow-up questions to promote a more thorough understanding of the patient's symptom experiences. In a study of male combat veterans, the authors found sensitivity of .96 and specificity of .80, suggesting sound performance.

PTSD Symptom Scale—Interview

Developed by Foa, Riggs, Dancu, and Rothbaum (1993), the PTSD Symptom Scale—Interview (PSS-I) possesses many strong clinical features that warrant its consideration for clinical and research use. Consisting of the 17 criteria for the PTSD diagnosis, the PSS-I uses Likert-type rating scales for each of the criterion symptoms. It can be scored as a continuous and dichotomous measure of PTSD and takes approximately 20 minutes for completion. Administering this measure to 118 women with sexual assault histories, Foa, Riggs, et al. (1993) found excellent interrater reliability, diagnostic sensitivity of .88, and specificity of .96. Test–retest reliability over 1 month was also reported to be strong.

The advantages of the PSS-I are its relative brevity, its promising psychometric properties, and its use of Likert rating scales that provide both a dichotomous and a continuous scoring routine. Another strength of this interview is its development and validation with sexual assault survivors, a population of great interest and clinical importance.

Self-Report PTSD Questionnaires

Several self-report measures have been developed as time- and cost-efficient ways of obtaining information about PTSD symptomatology. These measures enjoy widespread acceptance and use due to ease of administration and scoring, and they are also useful adjuncts to the structured diagnostic instruments. They can also be invaluable when used as screens

for PTSD. These measures are most frequently used as continuous measures of PTSD, but specific cutoff scores can be used in order to arrive at a diagnosis of PTSD.

PTSD Checklist

Developed by researchers at the National Center for PTSD in Boston, the PTSD Checklist (PCL) comes in two versions; one is for civilians, whereas the other is for military personnel. The scale contains the 17 items derived from the DSM diagnostic criteria, each scored on a 5-point Likert scale. Weathers, Litz, Herman, Huska, and Keane (1993) examined its psychometric properties and found excellent internal consistency (alpha = .97), excellent test–retest reliability over a 2- to 3-day period (.96), and strong correlations with other measures of PTSD. The association with the Mississippi Scale was .93, with the Keane PTSD Scale of the MMPI-2 was .77, and with the Impact of Event Scale (IES; see below) was .90. Blanchard, Jones-Alexander, Buckley, and Forneris (1996) used the PCL in their studies of motor vehicle accident survivors; they found that the correlation of the PCL with the CAPS was .93, and that its overall diagnostic efficiency was .90 when compared to the CAPS. The properties of the PCL with other populations have yet to be reported in the literature.

Impact of Event Scale

Initially developed by Horowitz, Wilner, and Alvarez (1979), the IES was revised by Weiss and Marmar (IES-R; 1997) to incorporate the symptoms of hyperarousal for PTSD (Criterion D). The original scale contained only reexperiencing symptoms and avoidance/numbing symptoms, and needed to be revised in order to parallel the diagnostic picture more closely. Although the authors have provided some preliminary data, more information is needed about the revision's reliability and validity. The original IES was the most frequently used measure of PTSD, and it possessed good psychometric properties. Similar studies with the IES-R will insure its continued use in clinics and research settings.

Mississippi Scale for Combat-Related PTSD

The Mississippi Scale (Keane et al., 1988) is a 35-item scale designed to measure combat-related PTSD. The items were selected from an initial pool of 200 items generated by experts to closely match the DSM-III criteria for the disorder. The Mississippi Scale has excellent psychometric properties, with an alpha of .94 and test–retest reliability of .97 over a 1-week interval. With a cutoff score of 107, the Mississippi Scale had strong sensitivity (.93) and specificity (.89).

These results were replicated in an independent laboratory by McFall, Smith, Mackay, and Tarver (1990), who found that the Mississippi Scale was highly correlated with the SCID PTSD module. These findings suggest that the Mississippi Scale, which is widely used in clinical and research settings serving veterans, is a valuable self-report tool.

Keane PTSD Scale of the MMPI-2

Originally derived from the MMPI Form R, the Keane PTSD Scale now consists of 46 items empirically drawn from the MMPI-2 (Keane, Malloy, & Fairbank, 1984; Lyons & Keane, 1992). The original report on the scale indicated that this Scale correctly classified some 82% of the 200 subjects in the study. Subsequent studies have confirmed these findings in combat veteran populations (Watson, Kucala, & Manifold 1986).

In terms of reliability, Graham (1990) found the Keane PTSD Scale to have strong internal consistency (.85–.87) and test–retest reliability (.86–.89). Although only a few studies have been conducted to date on this scale in nonveteran populations, the data presented appear to be promising (Koretzky & Peck, 1990). More research is needed in this area, especially in the area of forensic psychology, where the MMPI-2 is frequently employed because of its validity indices.

Penn Inventory for Posttraumatic Stress

The Penn Inventory is a 26-item questionnaire developed by Hammerberg (1992). Its psychometric properties have been examined in multiple trauma populations, and its specificity is comparable to that of the Mississippi Scale, while its sensitivity is only slightly lower. It has been used with accident survivors, veterans, and general psychiatric patients. It has been primarily employed with samples of male patients.

Posttraumatic Diagnostic Scale

Developed by Foa, Cashman, Jaycox, and Perry (1997), the Posttraumatic Diagnostic Scale (PTDS) is derived from the DSM criteria directly. The PTDS begins with a 12-question checklist to elucidate the traumatic events to which an individual might have been exposed. Next, the patient is asked to indicate which of the events experienced has bothered him or her the most in the past month. The patient then rates his or her reactions to the event at the time of its occurrence, in order to determine whether the event fits both Criterion A1 and Criterion A2. The patient next rates on a single 4-point scale the intensity *and* frequency of each of the 17 symptoms of PTSD he or she has experienced in the past 30 days. The final section of the scale asks for self-ratings of impairment across nine areas of life functioning.

The PTDS was validated on several populations, including combat veterans, accident survivors, sexual and nonsexual assault survivors, and persons experiencing other traumatic events. The psychometric analyses proved to be exceptional. For internal consistency, the coefficient alpha was .92 overall; test–retest reliability for the diagnosis of PTSD over a 2- to 3-week interval was also high (kappa = .74). For symptom severity, the test–retest correlation was .83. When the PTDS was compared to the SCID PTSD module, a kappa coefficient of .65 was obtained with 82% agreement; the sensitivity of the test was .89, whereas its specificity was .75. Clearly, this self-report scale functioned well in comparison to the clinician ratings obtained in the SCID. It is a useful self-report and screening device for measuring PTSD and its symptom components.

The use of these self-report questionnaires in a wide range of clinical and research contexts seems well supported by the extant data. It is clear that they can be successfully employed to measure PTSD symptoms when administering a structured diagnostic interview is not feasible or practical. Many of the measures can be used interchangeably, as the findings appear to be robust for the minor variations in methods and approaches involved. In selecting a particular instrument, the clinician is encouraged to examine the data for that instrument for the population on which it is to be employed. In so doing, the clinician is apt to maximize the accuracy and efficiency of the chosen test.

Psychophysiological Measures

Research on biologically based measures of PTSD has grown tremendously in the past 10 years. Findings suggest that PTSD alters a wide range of physiological functions (Yehuda,

1997) and may also affect structural components of the brain (particularly the hippocampus; Bremner et al., 1995; see Chapter 6). To date, these findings have not been subjected to rigorous psychometric testing (i.e. utility analyses) to determine the extent to which these deviations are predictive of PTSD and non-PTSD cases. The primary exceptions to this conclusion are findings in the area of psychophysiological reactivity, which from the start examined diagnostic accuracy (e.g. Blanchard, Kolb, Pallmeyer, & Gerardi, 1982; Malloy, Fairbank, & Keane, 1983; Pitman, Orr, Forgue, de Jong, & Claiborn, 1987).

The findings in this area clearly point to the capacity of psychophysiological indices to identify and classify cases of PTSD on the basis of reactivity to audio, audiovisual, and imagery-based cues. Measures have included heart rate, blood pressure, skin conductance, and electromyography. Studies covered the range of traumatic events and included motor vehicle accident survivors, combat veterans from available eras, female sexual assault survivors, and survivors of terrorism. In perhaps the largest study of its kind, Keane et al. (1998) examined the responses of over 1,000 combat veterans to audiovisual and imagery-based cues of combat experiences. The results supported the presence of elevated psychophysiological arousal and reactivity in the participants, more than two-thirds of whom were correctly classified as having or not having PTSD.

Clearly, psychophysiological assessment is expensive in terms of time, patient burden, and cost. Yet, in cases where much is at stake, it may be helpful to employ this assessment strategy clinically (cf. Prins, Kaloupek, & Keane, 1995). Widespread adoption of this method of assessment is not anticipated, however, due to the costs, the expertise required, and the success of more economical methods of assessment (such as the diagnostic interviews and the psychological tests that are available).

As more information is collected on measures of the hypothalamic–pituitary–adrenocortical axis, it is indeed possible that this system and measures of it could be useful adjuncts to the diagnosis of PTSD as well as other anxiety and mood disorders. In particular, indices of cortisol, norepinephrine, and their ratio appear ready for an intensive examination for their capacity to improve diagnostic hit rates for PTSD, above and beyond the use of diagnostic interviews and psychological tests (Yehuda, Giller, Levengood, Southwick, & Siever, 1995); however, questions of specificity relative to other anxiety disorders are likely to prove formidable.

Summary of Findings on Assessment

Diagnosing and assessing outcomes in PTSD constitute a topic of growing interest and concern in the mental health field (Wilson & Keane, 1997). Since the inclusion of PTSD in DSM-III, there has been considerable progress in understanding and evaluating the psychological consequences of exposure to traumatic events. Conceptual models of PTSD assessment have evolved (Keane et al., 1987; Sutker, Uddo-Crane, & Allain, 1991); psychological tests have been developed (Foa et al., 1997; Norris & Riad, 1997); diagnostic interviews have been validated (Davidson et al., 1989; Foa, Riggs, et al., 1993; Weathers et al., 1992); and subscales of existing tests have been created to assess PTSD (e.g., MMPI-2, Keane et al., 1984; Symptom Checklist 90—Revised, Saunders, Arata, & Kilpatrick, 1990). We can rightly conclude that the assessment tools available to evaluate PTSD are comparable to or better than those available for any disorder in the DSM. Multiple instruments have been developed to cover the range of needs of the clinician. The data on these instruments are nothing short of outstanding.

Clearly, the assessment of PTSD in clinic settings must focus on more than the presence–absence, and severity of PTSD. A comprehensive assessment strategy would certainly gather

information about an individual's family history, life context, symptoms, beliefs, strengths, weaknesses, support system, and coping abilities (Newman, Kaloupek, & Keane, 1996). This would assist in the development of an effective treatment plan for the patient. The primary purpose of the present review has been to examine the quality of a range of different instruments used to diagnose and assess PTSD; of course, the comprehensive assessment of a patient also needs to include indices of social and occupational functioning. Finally, a satisfactory assessment ultimately relies upon the clinical, and interpersonal skills of the clinician, since many topics related to trauma are inherently difficult for a patient to disclose to others.

This part of the chapter is not intended to be comprehensive in its review of the psychometric properties of all instruments available. Its goal has been to provide a heuristic structure that clinicians might employ when selecting a particular instrument for their clinical purposes. By carefully examining the psychometric properties of an instrument, the clinician can make an informed decision about the appropriateness of a particular instrument for the task at hand. Instruments that provide a full utility analysis (i.e., sensitivity, specificity, hit rate, etc.) do much to assist clinicians in making their final judgments. Furthermore, instruments that are developed and evaluated on multiple trauma populations, across genders, and with different racial, cultural, and age groups are highly desirable; these are objectives for future study.

TREATMENT

Historical Precedents for Current Psychological Treatments

From a historical perspective, the work of Janet (1889) and Freud (1936) has most influenced the treatment of PTSD. The objectives of each approach have much in common with contemporary models of treatment, including the cognitive-behavioral treatments of today. Thus current models of treatment owe a substantive debt to these pioneers. As presented by Fenichel (1945), there are two components to the psychoanalytic treatment of "traumatic neurosis": (1) attempts to quiet the high levels of anxiety and reactivity to the event; and (2) attempts to reconstruct the details of the event with the accompanying emotional reactions, to promote mastery over memories of the event. The contemporary treatments of anxiety management training or AMT (i.e., stress management and stress inoculation treatments) and exposure therapies parallel the two aspects of treatment proposed by Fenichel in his classic text on psychoanalysis.

Janet's contributions, reanalyzed and discussed frequently since the inclusion of PTSD in DSM-III, focus on the phenomenology of symptoms often observed in traumatized patients. Janet was an early proponent of hypnosis and advocated its use in accessing the details of traumatic events. His recognition that a memory of a traumatic events is often fragmented and focused on particular aspects of the event at the expense of a more complete memory is one of his major contributions to our understanding of this subject. The inclusion of hypnosis as a viable treatment for traumatized people added a systematic and technical approach to achieving the psychoanalytic objective of reconstructing details of the event in order to achieve mastery over the event.

In the aftermath of World War II, the use of sodium amytal interviews to help patients recreate and recall details of traumatic war events became increasingly widespread. Again, the objective of these interviews was to identify critical elements of traumatic war events that were not reported by the patients, so that efforts to address the psychological

sequelae of these events could begin. Typically, the events involved human devastation, atrocities, terror in the face of death and danger, and helplessness.

The issue of traumatic memories has become controversial in the contemporary study of PTSD. Some deny the idea that memories of traumatic events have distinctive characteristics in comparison to memories of other life events; some challenge the nature of the constructs often invoked to explain traumatic memories (e.g. dissociation or repression); others are vigorously holding to scientific standards, recognizing that work in this area has largely only attained the scientific status of clinical observation. Despite the debate, it is clear that thousands of clinicians over the lengthy history of psychotherapy have noted that during therapy traumatized patients do indeed come to understand the nature and details of past traumatic events, whether these events occurred in childhood (as in the case of incest) or in adulthood (as with war veterans). Additional research directly on the issue of traumatic memory would contribute immeasurably to our understanding of developmental psychopathology and to the treatment of PTSD. Roth and Friedman (1998) assembled a group of notable scholars to summarize the literature on memories of trauma on behalf of the International Society for Traumatic Stress Studies. Their report delineated the issues, examined the extant evidence on traumatic memories, and outlined the needs for future scientific study of traumatic memory. The full text of this report is available on the society's Web site (www.istss.org).

Current Psychological Treatments

Largely driven by the psychological and social problems of returning American Vietnam veterans, mental health practitioners established treatment programs to treat traumatic disorders for these large numbers of war veterans (e.g., Keane & Kaloupek, 1982). Concomitantly, clinical researchers began to understand more fully the psychological consequences of exposure to sexual assault and rape (Burgess & Holmstrom, 1974; Kilpatrick, Veronen, & Resick, 1979), and to develop treatments for these problems.

At the outset, conceptual models of PTSD borrowed from Mowrer's (1960) two-factor learning theory (Fairbank & Keane, 1982; Keane, Zimering, & Caddell, 1985; Kilpatrick, Resick, & Veronen, 1981), which posits that fear and other aversive emotions are learned through association via classical conditioning mechanisms. This is the first factor in the acquisition of aversive emotions. The second factor is that individuals will do whatever is necessary to escape from and to avoid cues that stimulate these aversive emotions.

Theoretical models have since increasingly accommodated cognitive factors (Brewin, Dalgleish, & Joseph, 1996; Ehlers & Clark, 2000; Foa, Steketee, & Rothbaum, 1989; Lang, 1977a; Litz & Keane, 1989; see Chapters 3, 7, and 8). Treatments deriving from the behavioral and the cognitive models are among the most widely recommended and evaluated today. Specifically, exposure therapies and AMT techniques have evolved as two of the most widely accepted treatment approaches for patients with PTSD. These cognitive-behavioral treatments are also the most likely to be empirically tested by their proponents.

Although group therapies, both structured and unstructured, and psychodynamically informed psychotherapy are also widely used to treat PTSD, few studies in the literature have documented the clinical efficacy of these approaches (Foa, Keane, & Friedman, 2000). The studies that do exist do not meet contemporary standards for strong evidence. For example, most studies do not involve the use of randomized controlled clinical trials, which are the standard means of evaluating treatments throughout health and mental health care. However, the parallels between the objectives of psychoanalytic approaches as delineated

above and those of AMT and exposure therapies are unmistakable (i.e., directly reducing anxiety to cues of the event and mastering the memory).

A more recent approach to treating PTSD is eye movement desensitization and reprocessing (EMDR; Shapiro, 1989). This is essentially an atheoretical technique that was accidentally discovered to alter disturbing thoughts, feelings, and images (Shapiro, 1995). It has been applied to a broad range of psychological problems, but the bulk of the available research on its efficacy is with PTSD, and so it is discussed in this chapter.

Accordingly, this section on current psychological treatments for PTSD examines the data supporting the use of exposure therapies, AMT, and their combination with cognitive therapies, as well as EMDR. Other treatments for PTSD are in developing phases and, though important, do not yet have an empirical base (e.g., group therapies, interpersonal psychotherapy, psychodynamic therapy, acceptance and commitment therapy). Accordingly, these are not discussed at any length.

Exposure Therapies

There is a long, rich tradition for treating anxiety disorders with one or another of the exposure therapies, whether it be systematic desensitization, flooding, prolonged exposure, implosive therapy, or another form (e.g., Barlow, 1988; Levis, 1980; Rachman, 1980). Exposure therapy is applied to PTSD in two fundamental ways: by using *in vivo* exposure or imaginal exposure. *In vivo* exposure generally involves returning to the site of the traumatic event in order to reduce avoidance and promote mastery over the cues associated with the event. For example, if a person developed PTSD following a car accident, the therapist might consider returning to the scene of the accident with the patient during the course of a treatment regimen (Blanchard & Hickling, 1997).

Imaginal exposure is often used in treating PTSD when *in vivo* exposure isn't possible (e.g., returning to a distant location is impractical, returning to an earlier time in life isn't feasible). Cues are presented in imagery in an effort to describe details of an event or set of events from the perspective of the stimulus propositions, response propositions, and meaning propositions associated with the event (Lang, 1977a). Like *in vivo* exposure, imaginal exposure has as its goal the reduction of avoidance and the promotion of mastery. Concomitantly, in some cases it promotes recall of details of the traumatic event that were previously not reported. This is probably a result of the systematic use of the cues as reminders.

Keane and his colleagues were the first to apply exposure therapy to the treatment of PTSD. Initially, this application took the form of single-subject designed studies to document the effects of exposure to memories of the traumatic combat events experienced by veteran patients (Black & Keane, 1982; Fairbank & Keane, 1982; Keane & Kaloupek, 1982). Significant reductions in trauma symptoms, anxiety, and other related symptoms were noted as a function of these interventions. These case demonstrations were instructive because they employed a consistent conceptual model of PTSD in the implementation of a treatment; utilized systematic diagnostic measures for case identification; and measured outcome in replicable, standardized ways. Several of these cases also employed scientifically validated single-subject research methodologies (see Fairbank & Keane, 1982).

COMPARISONS AND COMBINATIONS WITH OTHER TREATMENTS. This preliminary work led to the development of a randomized clinical trial that compared two active treatments (imagery-based exposure therapy and AMT) to a wait-list condition in the treatment of combat-related PTSD in Vietnam veterans. The results of this study were clear-cut. Com-

pared to the wait-list condition, those patients receiving imagery-based exposure therapy showed reductions on standard psychometric instruments and on clinician ratings of symptoms at the posttreatment assessment. Changes on these measures were maintained at a 6-month follow-up evaluation. Interestingly, the less intensive AMT resulted in so many treatment dropouts that data were not available for analyses (Keane, Fairbank, Caddell, & Zimering, 1989).

Brom, Kleber, and Defares (1989) also conducted a randomized clinical trial comparing three active treatments for 112 patients who had experienced a traumatic event and were seeking psychotherapy. The treatments were exposure therapy (i.e., systematic desensitization), hypnotherapy, and psychodynamic treatment. Patients were evaluated before treatment, immediately after treatment, and at a 3-month follow-up with standardized psychometric instruments. The patients receiving exposure therapy showed a reduction in symptoms at posttest that was maintained at the 3-month follow-up. While comparable levels of change were noted in the other two active treatment groups, all three groups demonstrated greater change than the wait-list condition.

Foa, Rothbaum, Riggs, and Murdoch (1991) examined exposure therapy, an AMT condition called "stress inoculation," supportive counseling, and a wait-list condition in the treatment of rape-induced PTSD. Measures included clinical ratings of symptoms and standardized psychometric inventories, all administered at pretreatment, posttreatment, and a 3.5-month follow-up. The stress inoculation treatment was superior to the counseling and wait-list conditions at posttest. However, at the follow-up, the patients receiving exposure therapy performed better on measures of PTSD than patients in the other conditions did.

Three additional studies with combat veterans with PTSD demonstrate the salutary effects of exposure therapy. First, Boudewyns and Hyer (1990) and Cooper and Clum (1989) demonstrated that the addition of exposure therapy to available treatments of PTSD improved outcome for patients. Next, Glynn et al. (1999) assessed the effects of adding a family-based skills training intervention to imaginal exposure therapy. Comparing exposure therapy with behavioral family therapy to exposure therapy alone and to a wait-list comparison group, they found that the exposure therapy resulted in significant declines in symptoms of anxiety, arousal, and reliving the traumatic event, but did not affect the avoidant or numbing symptoms of the disorder. These treatment gains were maintained at the 6-month follow-up. Importantly, the addition of 16 sessions of family behavioral treatment did not yield incremental treatment gains.

Other combinations of treatment also appear to have promise in the amelioration of PTSD symptomatology. For example, Frueh, Turner, Beidel, Mirabella, and Jones (1996) assembled a multicomponent treatment for combat-related PTSD. These investigators combined exposure therapy, AMT, and cognitive therapy into a package for treating war veterans. They found positive treatment effects in a preliminary clinical trial that awaits more rigorous scientific review. Other packages combining these three forms of therapy are discussed later.

More recently, studies have appeared in the literature examining the comparative effectiveness of various approaches to treatment, including exposure therapy. For example, Tarrier et al. (1999) examined exposure therapy and cognitive therapy in the treatment of outpatients with PTSD stemming from many different traumatic events. Seventy-two patients entered treatment, and 62 patients completed treatment. Both groups manifested significant improvement that was maintained at the 6-month follow-up period. Outcomes were favorable for both groups, although there was no difference between the two at conventional levels of significance. Unfortunately, there was no attention control or wait-list control against which these two active treatments could be compared.

Similarly, Foa, Dancu, et al. (1999) compared exposure therapy to AMT and then combined the two treatments. These three groups were compared to a wait-list control group. All three of these treatments effectively reduced symptoms of rape-related PTSD and resulted in functional improvement. There were no differences among the three treatment groups on outcome measures, but all three groups improved more than the wait-list comparison group did.

In a study that once again compared exposure therapy to cognitive therapy, Marks, Lovell, Noshirvani, Livanou, and Thrasher (1998) examined these two treatments alone and in combination in outpatients with PTSD secondary to a wide range of traumatic events. A relaxation therapy condition was employed as the primary comparison group. All three active treatment conditions showed significant improvement, and greater improvement than that observed in the relaxation group. The three active treatments did not differ from one another on the key outcome variables.

Several investigations have advanced the field of PTSD treatment, even though the methodology utilized in the outcome study limited the conclusions that could be drawn. Frank and Stewart (1983) reported the effects of systematic desensitization on women who had been raped and who developed significant psychological symptomatology. Compared to an untreated comparison group, those women treated with graduated exposure improved most on a range of anxiety and depression symptom measures.

Richards, Lovell, and Marks (1994) compared imaginal and *in vivo* exposure in a randomized study of survivors of diverse traumatic events. At the 12-month follow-up, patients reported consistent reductions in PTSD symptoms and improved social adjustment. These data further substantiate the effectiveness of exposure therapy for some patients, and also suggest that improvements in symptoms are also reflected in critical domains of life functioning. In summary, the extant data support the use of exposure therapy in the treatment of PTSD. In a previous review of this literature, Solomon, Gerrity, and Muff (1992) derived the same conclusion from data available at that time. Similar conclusions were drawn by Otto, Penava, Pollack, and Smoller (1996) in a more recent review of the literature.

As data continue to accrue on exposure therapy, there is a distinct need for studies to examine combinations of treatments, to employ measures that assess social and occupational functioning, and to address the impact of treatments on comorbid psychological conditions. Clearly, the available efficacy studies demonstrate the value of extending the use of exposure therapies to patients with PTSD. Because their rich tradition is deeply rooted in experimental psychology and has been tested in the treatment of many anxiety disorders, exposure therapy in its many formats should be given priority by clinicians encountering patients with PTSD. Future studies assessing the generalization of exposure therapy from laboratory trials (efficacy studies) to clinical settings (effectiveness studies) would be particularly welcome.

EXPOSURE THERAPY IN THE PREVENTION OF PTSD. In what may ultimately prove to be an important lesson for the treatment of individuals exposed to traumatic events, Foa, Hearst-Ikeda, and Perry (1995) examined the efficacy of a brief intervention to prevent the development of chronic PTSD. For women who had been recently raped, the authors developed a program based upon that which worked so well in earlier trials with chronic PTSD. Exposure therapy figured prominently in the package of treatments assembled. This package also included elements of education, breathing retraining, and cognitive restructuring. When patients receiving the package were compared to a matched control group, this study found that at 2 months after intervention only 10% of the treated group met criteria for PTSD, while 70% of the untreated comparison group did.

Similarly, Bryant, Harvey, Dang, Sackville, and Basten (1998) constructed a treatment package consisting of exposure therapy, cognitive restructuring, and AMT in the treatment of acute stress disorder, a disorder that follows exposure to traumatic events but precedes the development of PTSD (i.e., it occurs within a month of exposure). In a randomized controlled trial, they found that individuals provided with the five sessions of this package as compared to supportive counseling met criteria for PTSD less often (17%) than did those receiving supportive counseling (67%) at a 6-month follow-up evaluation.

Anxiety Management Training

Typically, AMT involves teaching patients an assortment of behavioral and cognitive strategies to enhance their capacity to manage the emotions associated with PTSD. Such skills may include relaxation training, breathing retraining, trauma education, guided self-dialogue, cognitive restructuring, and communication skills training. Some programs for PTSD have emphasized the incorporation of anger management training as a part of the skills taught to patients (Chemtob, Novaco, Hamada, & Gross, 1997; Keane et al., 1989), given the salience of this interpersonal problem among patients with PTSD.

The studies described above by Foa and colleagues (Foa, Rothbaum, et al., 1991; Foa, Dancu, et al, 1999) and Keane et al. (1989) compared exposure therapy to AMT. In the Keane et al. study, therapists were instructed to explicitly avoid discussing or processing the traumatic events of the patients, in an effort to minimize the amount of exposure provided in this treatment condition. Perhaps this severe restriction led to the high rates of dropout in the AMT condition. Although the treatment appeared to be face-valid in its emphasis on treating the precise symptoms of PTSD, it evidently didn't provide sufficient relief to the combat veterans enrolled in this trial. However, this treatment did result in significant reductions in symptoms for female rape victims in both of Foa and colleagues' studies. The long-term effects were just not as strong as those found for exposure therapy.

Some studies primarily employed one form of AMT, rather than a multifaceted treatment package. For example, Peniston (1986) completed a project examining the effects of biofeedback-assisted relaxation treatment for veterans with combat-related PTSD. This form of AMT did result in significant short-term positive effects for the experimental group, although the long-term effects of this intervention were not systematically investigated.

Similarly, Chemtob et al. (1997) presented data on the treatment of anger and rage in veterans with PTSD, using a randomized controlled clinical design. Although the sample size of this study was small, behavioral treatment employing an anger-focused version of AMT yielded impressive reductions on psychometric measures of anger and on laboratory measures (behavioral) of anger reactivity. Importantly, these changes were associated with reductions in the reexperiencing symptoms of PTSD.

Clearly, there is evidence to suggest that a skills training approach such as AMT can have a favorable impact on symptoms of PTSD. Although the data are neither as strong nor as consistent as those for exposure therapies, it seems reasonable to conclude that there is some empirical foundation for the use of AMT in treating PTSD. An application with possibly the strongest potential for treating PTSD is a combination of various forms of therapy that includes exposure therapy, cognitive therapy, and AMT.

Combinations of Exposure Therapy, Cognitive Therapy, and AMT

Resick and Schnicke (1992) have proffered a multidimensional behavioral treatment package for women who have rape-related PTSD. This package, entitled "cognitive processing

therapy" (CPT), combines elements of exposure therapy, AMT, and cognitive restructuring. The cognitive therapy component of CPT involves addressing key cognitive distortions found among women who have been assaulted. In particular, these authors have designed interventions for addressing difficulties in safety, trust, power, self-esteem, and intimacy in the lives of survivors. In a preliminary evaluation of CPT, the authors compared outcomes at pretreatment, posttreatment, 3 months, and 6 months for a treatment group and a wait-list comparison group (no random assignment was used). On clinician ratings and psychometric inventories of PTSD, the patients receiving CPT improved markedly. At the posttreatment assessment, impressively, none of the treated patients met criteria for PTSD.

In a recently completed study, Resick, Nishith, and Astin (2000) reported on a comparison of CPT and exposure therapy in the treatment of rape-related PTSD. In general, the two treatments were equally effective and more effective than a wait-list control condition. CPT did also seem to reduce comorbid symptoms of depression, as well as those of PTSD.

Combination treatments that include an array of cognitive-behavioral strategies have the advantage of addressing multiple problems that people with PTSD may exhibit, as well as incorporating techniques that have considerable empirical support in the clinical literature. Keane, Fisher, Krinsley, and Niles (1994) described a treatment package including exposure therapy, AMT, and cognitive restructuring as central features of their approach to treating PTSD. This package employs a phase-oriented approach to treating severe and chronic PTSD that includes the following six phases: (1) behavioral stabilization; (2) trauma education; (3) AMT; (4) trauma focus work; (5) relapse prevention skills; and (6) aftercare procedures (see Table 12.1).

Although this approach has intuitive clinical appeal, it wasn't until psychologists Fecteau and Nicki (1999) examined such a package in a randomized clinical trial for PTSD secondary to motor vehicle accidents that the impact of a combination package such as that proposed by Keane et al. (1994) was assessed. Their intervention consisted of trauma education, relaxation training, exposure therapy, cognitive restructuring, and guided behavioral practice. Patients were randomly assigned to the intervention or to a wait-list comparison group and received some 8–10 sessions of individualized treatment. The results of the intervention were successful as measured by clinical ratings, self-report questionnaires, and a laboratory-based psychophysiological assessment procedure. Described by the authors as clinically as well as statistically significant, these treatment effects were maintained at the 6-month follow-up assessment.

Thus there appear to be at least three treatments with excellent empirical support for treating PTSD: exposure therapy, AMT, and cognitive therapy. These three approaches have excellent empirical support in well-controlled clinical trials (efficacy studies), manifest strong treatment effect sizes, and appear to work well across diverse populations of trauma survivors. Future studies to examine the effectiveness of these approaches in clinic settings (effectiveness studies) are warranted.

Eye Movement Desensitization and Reprocessing

EMDR is a technique designed by Shapiro (1989, 1995) that has received considerable attention from practitioners and academics alike. Worldwide training institutes are well attended by clinicians seeking to learn about EMDR and its use in PTSD. Although Shapiro (1995) alleges that this technique is helpful for treating a range of disorders, its use is often directly associated with PTSD.

TABLE 12.1. A Phase-Oriented Approach to the Cognitive-Behavioral Treatment of PTSD

Phase 1: Behavioral stabilization
- Alcohol and drug use addressed.
- Basic needs of food, shelter, and safety insured.
- Proximal crises precipitating treatment controlled.
- Acceptance of therapy and the therapeutic relationship.
- Pharmacological assistance considered to improve emotional control.

Phase 2: Trauma education
- Reactions, responses, and symptoms that follow trauma exposure explained.
- Patient's responses normalized.
- Common interpersonal, marital, and vocational effects explored.
- Physiological signs and symptoms reviewed.
- Anger as a counterconditioning agent to anxiety explained.

Phase 3: Anxiety management training (AMT)
- Breathing retraining taught.
- General relaxation training considered for patient.
- Identify those cognitive beliefs and distortions contributing to distress.
- Teach rational self-statements.
- Communication skills training surrounding the traumatic experience.
- Anger management skills considered.

Phase 4: Trauma focus work
- Systematic desensitization using AMT skills.
- Prolonged exposure to traumatic memories.
- Structured group therapy exposure sessions.
- Use of imagery and *in vivo* therapy and homework assignments to promote mastery and reduce avoidance.
- Systematic uncovering work.

Phase 5: Relapse prevention
- Management of trauma cues and anniversary reactions.
- Education about substance use when cues are triggered.
- Identifying and managing situations where control is compromised.
- Mobilization of social support systems.
- Teaching the use of appropriate communication skills to seek assistance when needed.
- Attention to the need for intimate relationships.

Phase 6: Aftercare services
- Use of community-based support, such as Alcoholics Anonymous and survivor groups.
- Community involvement and social activism to address the needs of other survivors.
- Teaching appropriate skills to access professional care when needed.

In the development of EMDR, Shapiro (1989) conducted a series of single-subject cases and open clinical trials, which suggested that this approach to treating the psychological effects of trauma exposure may promote recovery. From an operational perspective, the fundamentals of EMDR are (1) the evocation of trauma-relevant images and memories, (2) the psychological evaluation of the aversive qualities of these images/memories, (3) the identification (with or without therapist assistance) of an alternative cognitive appraisal of the image/memory, (4) examination of physiological reactions to the image/memory, (5) focusing on the idiographically determined positive appraisal of the image/memory, and (6) repeated sets of lateral eye movements while the patient is focusing on elements of the traumatic response or the alternative cognitive appraisal.

Examining the efficacy and effectiveness of EMDR is challenging. Its mechanism of action is not based on any contemporary theories of human behavior, learning, or cogni-

tive science, although some discussion of Pavlovian neurophysiology is provided across publications (Shapiro, 1989, 1995). For this reason, it has been seriously criticized in the scientific literature (Herbert & Mueser, 1992; Keane, 1998; Lohr, Kleinknecht, Tolin, & Barrett, 1995; Lohr, Tolin, & Lilienfeld, 1998). Yet the absence of theory or a conceptual foundation is not sufficient to enable us to dismiss totally the preliminary findings on this technique. If there is efficacy, then serious scholars can assume responsibility for identifying the precise mechanism responsible for any effects observed. The question is therefore "Is there evidence for EMDR's efficacy?", and, more importantly, "Is EMDR more effective or efficient than tested techniques?"

Carlson, Chemtob, Rusnak, Hedlund, and Muraoka (1998) compared EMDR to biofeedback-assisted relaxation training and to routine clinical care. In this randomized clinical trial with combat veterans, the authors reported that at the 180-day follow-up, the group receiving EMDR showed greater clinical improvement than either of the two comparison groups on self-report, psychometric, and clinician-rated measures of PTSD.

Wilson, Becker, and Tinker (1995) reported on the treatment of traumatic memories in a heterogeneous sample of individuals recruited through newspaper advertisements and other means. Half of their subjects received EMDR, while the remaining half were placed on a waiting list. Of the 80 subjects in this study, fewer than half reached DSM criteria for PTSD. Following three 90-minute sessions of EMDR, the treated group demonstrated a greater reduction on psychometric measures and clinician ratings of symptoms than did the wait-list subjects.

Rothbaum (1997) utilized EMDR in a sample of female rape survivors with PTSD. She found strong treatment effects when the active treatment was compared to a wait-list comparison group. Similarly, Chemtob, Nakashima, Hamada, and Carlson (in press) found that EMDR was an effective treatment for PTSD in children who survived Hurricane Iniki on the island of Kauai in the state of Hawaii. This study also compared EMDR to a wait-list comparison group.

In an HMO setting, Marcus, Marquis, and Sakai (1997) found that EMDR was more effective than standard psychological care among individuals with PTSD due to diverse traumatic events. These findings are impressive because the study was conducted in a clinical setting, where control over the patient characteristics and therapist behavior (particularly for the experimental treatment) is difficult to attain.

Devilly, Spence, and Rapee (1998) compared EMDR to a similar type of treatment but without the eye movements in the treatment of combat-related PTSD in Australian Vietnam veterans. Each of these treatments was also compared to a supportive treatment comparison. The results of this study indicated that both active treatment groups improved equally; there was no apparent benefit to the inclusion in this treatment package of the eye movements per se. Pitman et al. (1996) also found no differences between EMDR with the eye movements and EMDR without the eye movements in the treatment of combat-related PTSD among American Vietnam veterans. These studies contribute to the growing skepticism about the value of the eye movement processes in achieving the outcomes observed to date in using EMDR with PTSD.

Other studies on EMDR's efficacy have been less encouraging. Projects by Boudewyns, Stwertka, Hyer, Albrecht, and Sperr (1993), Jensen (1994), and Vaughan et al. (1994) found only modest effects for EMDR. These studies all have significant methodological limitations, but they are comparable in quality and design to many of those projects providing the empirical support for this technique. One study by Renfrey and Spates (1994) found no differences between a group that received the eye movements and a similar group that didn't, contributing to the mounting evidence challenging their role in the treatment.

In summary, much work needs to be done before research will firmly support the use of EMDR in the treatment of PTSD. To date, there is not a single study supporting EMDR (as espoused by its developer) as a treatment superior to any of the existing treatments for PTSD, such as exposure therapy, AMT, or cognitive therapy. Such studies would provide needed evidence in the debate about the ultimate contribution of EMDR to the scientific and clinical literature (Chemtob, Tolin, van der Kolk, & Pitman, 2000; Lohr et al., 1998). Unlike the various forms of exposure therapy, which have a long tradition of ameliorating a range of anxiety-mediated clinical problems and which are embedded in the rich conceptual tradition of experimental psychology, EMDR falters seriously at the theoretical level. Needed are basic studies to examine the effects of eye movements (or other laterally alternating stimuli); small-scale, well-controlled efficacy studies that meet contemporary standards for treatment outcome research; and the formulation of a testable theory for the technique. Implicit in this latter recommendation are the development of a conceptual model of PTSD, and an explanation of how EMDR attempts to correct either the deficits or excesses involved in this disabling psychological condition.

To conclude the review of EMDR, it may be valuable to specify the possible strengths of this technique. First, EMDR does share some components of exposure therapy and cognitive therapy. These overlapping components should be identified and operationalized to promote our ability to study the approach more fully. Second, the technique builds assessment into the ongoing therapy process. Third, EMDR suggests that instructions to modify images and alter cognitive self-statements may be a reasonable treatment objective for patients with PTSD. Fourth, proponents of this technique have always noted the need for empirical documentation to support their approach.

If there is a fundamental weakness of EMDR, it stems from a distinct lack of integration with existing psychological models of psychopathology and psychotherapy. Although all existing models of PTSD clearly have their own limitations, it is incumbent upon the proponents of EMDR to hypothesize how their view of the problems associated with PTSD differs from others; how the theoretical mechanisms of their technique derive from behavioral theory and/or cognitive neuroscience; and how this technique functions to allay specific targeted symptoms or to create alternative cognitive structures of the traumatic event, and thus contributes to our understanding of this multidimensional disorder.

Psychopharmacological Treatments

Psychopharmacological treatments for PTSD are in the nascent stages of development. Although there have been numerous randomized placebo-controlled clinical trials across medication types, few studies have been replicated across research laboratories. Initial results of randomized clinical trials appeared to favor the use of the antidepressant amitriptyline (Davidson et al., 1990). More recently, two studies suggested that the selective serotonin reuptake inhibitor (SSRI) sertraline improved the symptom picture of patients with PTSD (Brady et al., 2000; Davidson, Landburg, et al., 1997). These patients (200 in each of the two trials) manifested significant improvement across clinician-rated and self-report outcome measures. Patients tolerated well the dosages (50–200 mg) utilized in the study; sleep problems (insomnia) were the primary complaints of the participants.

Similarly, van der Kolk et al. (1994) found fluoxetine to be effective in the treatment of PTSD in women. This randomized placebo-controlled clinical trial resulted in improvement in the numbing and arousal symptoms of PTSD, but little impact on the reliving or intrusive symptoms. Importantly, this trial also showed little effect on the PTSD symptoms of combat-related PTSD in males.

In these trials of the SSRIs the treatment effects were significantly smaller than those observed in the cognitive-behavioral treatment trials and in the psychotherapy trials more broadly. Clearly, there is a need for additional research on the neurobiology of PTSD, so that newer medications can be developed to target those neurobiological systems that experience dysregulation in PTSD. Virtually all neurobiological systems studied thus far are adversely affected by the presence of PTSD. Efforts to improve psychosocial functioning by indexing a single neurobiological system may be inadequate in the treatment of a disorder with this level of biological complexity.

Psychopharmacological treatment of PTSD is indeed in its earliest stages (Friedman, 2000). Yet there is some definite improvement in the symptom picture when the SSRIs are employed. It is also clear that relatively large treatment effects are found when patients are treated with cognitive-behavioral strategies. It seems logical, then, that trials combining SSRIs and cognitive-behavioral treatments should receive priority, to determine whether the interaction of these two approaches is superior to either one alone. With the use of contemporary methods of assessing outcome, it is likely that studies could detect both main effects and interaction effects that may improve psychological symptoms and psychosocial functioning in patients with PTSD.

In summary, the introduction of the SSRIs has spurred important new research on the treatment of PTSD. With the completion of several well-controlled, multisite clinical trials, and with the recent approval of sertraline for the treatment of PTSD by the Food and Drug Administration, the psychopharmacology of PTSD holds much promise for continuing and additional developments. Because PTSD is a disorder that affects as much as 8% of the general population, it is likely that pharmaceutical companies will continue to devote research and development efforts to finding more and more effective treatments for it. Clinical trials that examine the interactive effects of cognitive-behavioral treatments and psychopharmacological agents are clearly warranted in the next generation of treatment outcome studies.

Future Treatment Development

There is much to be learned about the treatment of PTSD. To be sure, there will be no simple answers for treating people who have experienced the most horrific events life offers. Undoubtedly, combinations of treatments as proposed by Keane et al. (1994) and Resick and Schnicke (1992) may prove to be the most powerful interventions.

As these treatment packages are developed, there is a need for additional work from perspectives other than cognitive-behavioral ones. Interpersonal psychotherapy (Klerman, Weissman, Rounsaville, & Chevron, 1984) and other short-term psychodynamically informed treatments need to be developed, evaluated, and then compared to existing behavioral and cognitive-behavioral treatments to determine which patients benefit most from these methods (Kudler, Blank, & Krupnick, 2000).

Similarly, there is a need to develop psychopharmacological interventions further so that they can be compared and contrasted with effective psychological methods, both individually and in combination. PTSD research in this area is only in the earliest stages of its development.

Finally, an assumption about the uniformity of traumatic events has been made in the literature in general (and throughout this chapter). Although it is reasonable to speculate that fundamental similarities exist among patients who have experienced diverse traumatic events and then develop PTSD, whether these patients will respond to clinical interventions in the same way is an empirical question that has yet to be addressed. Studies posing a

question such as this would be a welcome addition to the clinical literature: Will people with PTSD resulting from combat, torture, genocide, and natural disasters all improve as well as those treated successfully following rape, motor vehicle accidents, and assaults? This is a crucial issue that requires additional scientific study in order to provide clinicians with the requisite evidence supporting the use of available techniques.

Worldwide, the problems associated with war, rape, violence, criminal assault, and disaster do not appear to be declining. As a result, sound public policy is needed to guide society's response to survivors of these experiences. PTSD in its most chronic form is a debilitating condition that affects individuals, their families, their communities, and their nations. Those who are the targets of violence may ultimately become perpetrators, thus contributing to the cycle of violence initially documented by Widom (1989). If this is so, then interventions need to be implemented to prevent the occurrence of violence (primary prevention) or to mitigate its effects once it occurs (secondary prevention). Reliance upon sound empirical work to devise and implement these prevention efforts may ultimately be the best solution to the problems associated with PTSD.

FUTURE STUDIES

The evidence on the prevalence of exposure to traumatic events and the prevalence of PTSD is excellent in the United States. Yet there are fundamental errors in assuming that these prevalence rates apply even to other Western, developed countries. Studies that examine the prevalence of PTSD and other disorders internationally are clearly needed. Implicit in this recommendation is the need to examine the extent to which current assessment instrumentation is culturally sensitive to the ways in which traumatic reactions are expressed internationally. Much work on this topic will be required before definitive conclusions regarding prevalence rates of PTSD internationally can be drawn.

Studies of the effectiveness of the psychological and pharmacological treatments across cultures and ethnic groups are also needed. What may be effective for Western populations may be inadequate or possibly even unacceptable treatment for people who reside in other areas of the world and who have different world views, beliefs, and perspectives. This issue will need to be more closely examined before we can draw definitive conclusions. With the world's refugee population exploding, many of whom undoubtedly have PTSD, there is a clear need to test available treatments to see whether they can alleviate suffering in these diverse populations as these strategies have done in the United States.

Another area of increasing research importance, and one worthy of considerable energy and effort, is the impact of PTSD on health outcomes. In an important preliminary study, Felitti et al. (1998) examined a large sample of outpatients enrolled in the Kaiser Permanente HMO (n = 9,508). They found that as individuals experienced increasing numbers of adverse childhood experiences, they were increasingly at risk for a range of mental and physical health problems. These problems included disease risk factors such as obesity, alcohol use, drug use, cigarette smoking, and risky sexual behavior. When individuals who had experienced four or more adverse childhood experiences were examined, important relationships between the events and disease conditions themselves emerged: These people were more likely to develop diabetes, emphysema, stroke, cancer, and cardiovascular disease. These were statistically found to be direct effects of the exposure, operating above and beyond the indirect effects of engaging in high-risk behaviors.

The relationship among traumatic events, PTSD, and health outcomes is important for a wide range of reasons. Stress has been implicated in many different illnesses for more

than 100 years. Trauma exposure and PTSD provide an important arena for examining the interrelationship of stress and health. The level of stress and anxiety in PTSD is extreme, and the prevalence of the disorder in the population is high. It is distinctly likely that if we were to develop important leads in the interaction of stress and health, we might very well do so by examining those who have been exposed to traumatic life events. Currently the correlational evidence is strong that trauma exposure and PTSD result in increased service utilization, high-risk behavior, and even certain diseases (Schnurr, Friedman, Sengupta, Jankowski, & Holmes, 2000). The exact nature of the processes involved requires additional scientific inquiry. The possibility of unlocking the complex relationship among environmental events, stress, PTSD, and disease will challenge researchers at all levels of the analysis: cellular, organ, systemic, behavioral, and social. The prospect of addressing these issues successfully is among the most exciting enterprises facing clinical research today.